```
=> file biosis caba caplus lifesci medline
=> e coates anthony r/au
E1          13     COATES ANTHONY/AU
E2           7     COATES ANTHONY G/AU
E3           8 --> COATES ANTHONY R/AU
E4         105     COATES ANTHONY R M/AU
E5           7     COATES ANTHONY ROBERT MILNES/AU
E6           2     COATES ANTONY R M/AU
E7           5     COATES ARNOLD C/AU
E8           7     COATES ARTHUR D/AU
E9           3     COATES ARTHUR M/AU
E10          7     COATES ASHLEY/AU
E11          7     COATES ASHLEY O/AU
E12         16     COATES B/AU

=> s e3-e6 and ((heat shock)or(chaparoni?))
L1          35 ("COATES ANTHONY R"/AU OR "COATES ANTHONY R M"/AU OR "COATES
               ANTHONY ROBERT MILNES"/AU OR "COATES ANTONY R M"/AU) AND ((HEAT
               SHOCK) OR(CHAPARONI?))

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2           20 DUP REM L1 (15 DUPLICATES REMOVED)

=> d bib ab kwic 1-
YOU HAVE REQUESTED DATA FROM 20 ANSWERS - CONTINUE? Y/(N):y

L2   ANSWER 1 OF 20  BIOSIS  COPYRIGHT (c) 2008 The Thomson Corporation  on STN
     DUPLICATE 1
AN   2008:102620  BIOSIS <<LOGINID::20080330>>
DN   PREV200800097012
TI   Plasma   ***heat***     ***shock***   protein 60 and cardiovascular
     disease risk: the role of psychosocial, genetic, and biological factors.
AU   Shamaei-Tousi, Alireza; Steptoe, Andrew; O'Donnell, Katie; Palmen, Jutta;
     Stephens, Jeffrey W.; Hurel, Steven J.; Marmot, Michael; Homer, Karen;
     D'Aiuto, Francesco;   ***Coates, Anthony R. M.***  ; Humphries, Steve E.;
     Henderson, Brian [Reprint Author]
CS   Univ Coll London, Eastman Dent Inst, Div Microbial Dis, London WC1X 8LD,
     UK
     b.henderson@eastman.ucl.ac.uk
SO   Cell Stress & Chaperones, (WIN 2007) Vol. 12, No. 4, pp. 384-392.
     ISSN: 1355-8145.
DT   Article
LA   English
ED   Entered STN: 6 Feb 2008
     Last Updated on STN: 6 Feb 2008
AB   The Whitehall Study is a prospective epidemiological study of
     cardiovascular risk factors in healthy members of the British Civil
     Service, which has identified psychological distress as a major risk
     factor for coronary heart disease.  The levels of circulating Hsp60 in 860
     participants from the Whitehall cohort and 761 individuals diagnosed with
     diabetes have been measured and related to psychological, biological, and
     genetic factors.  In the Whitehall participants, concentrations of Hsp60
     ranged from undetectable to mg/mL levels.  Circulating Hsp60 correlated
     with total and low-density lipoprotein (LDL) cholesterol and was
     positively associated with a flattened slope of cortisol decline over the
     day.  Levels of this stress protein also correlated with measures of
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psychological stress including psychological distress, job demand, and low emotional support. Mass spectrometric analysis of circulating immunoreactive Hsp60 reveal that it is predominantly the intact protein with no mitochondrial import peptide, suggesting that this circulating protein emanates from mitochondria. The Hsp60 is stable when added to plasma and the levels in the circulation of individuals are remarkably constant over a 4-year period, suggesting plasma levels are partly genetically controlled. Sequence analysis of the HSP60-HSP10 intergenic promoter region identified a common variant 3175 C>G where the G allele had a frequency of 0.30 and was associated with higher Hsp60 levels in 761 type 2 diabetic patients. The extended range of plasma Hsp60 concentrations in the general population is genuine and is likely to be related to genetic, biological, and psychosocial risk factors for coronary artery disease.

TI Plasma ***heat*** ***shock*** protein 60 and cardiovascular disease risk: the role of psychosocial, genetic, and biological factors.

AU. . . Shamaei-Tousi, Alireza; Steptoe, Andrew; O'Donnell, Katie; Palmen, Jutta; Stephens, Jeffrey W.; Hurel, Steven J.; Marmot, Michael; Homer, Karen; D'Aiuto, Francesco; ***Coates, Anthony R. M.*** ; Humphries, Steve E.; Henderson, Brian [Reprint Author]

IT . . .
(MeSH)

IT Diseases
coronary heart disease: heart disease, genetics, CHD
Coronary Disease (MeSH)

IT Chemicals & Biochemicals
low-density lipoprotein cholesterol [LDL-C]; ***heat*** ***shock*** protein 60 [Hsp60]: expression, regulation

GEN human HSP60 gene [human ***heat*** ***shock*** protein 60 gene] (Hominidae): allele, expression; human HSP10 gene [human ***heat*** ***shock*** protein 10 gene] (Hominidae): allele, expression

L2 ANSWER 2 OF 20 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2008:37094 CAPLUS <<LOGINID::20080330>>

DN 148:189016

TI Differential regulation of circulating levels of molecular chaperones in patients undergoing treatment for periodontal disease

AU Shamaei-Tousi, Alireza; D'Aiuto, Francesco; Nibali, Luigi; Steptoe, Andrew; ***Coates, Anthony R. M.*** ; Parkar, Mohamed; Donos, Nikos; Henderson, Brian

CS Division of Microbial Diseases, UCL Eastman Dental Institute, University College London, London, UK

SO PLoS One (2007), 2(11), No pp. given
CODEN: POLNCL; ISSN: 1932-6203
URL: http://www.plosone.org/article/fetchObjectAttachment.action?uri=info%3Adoi%2F10.1371%2Fjournal.pone.0001198&representation=PDF

PB Public Library of Science

DT Journal; (online computer file)

LA English

AB Background. Evidence is emerging that mol. chaperones, in addn. to their intracellular protein folding actions, can act as intercellular signaling proteins with an ability to modulate leukocyte function. Recent evidence has also shown that these proteins can exist in the circulation and may be involved in disease pathogenesis. We have used periodontitis and its treatment as a model of inflammation in the human to det. its effects on levels of circulating HSP10, HSP60 and BiP. Methodol. / Principal Findings. A group of periodontal patients and matched controls were examd.

at the beginning of the study and then at 1 day and 6 mo following periodontal or control therapy. Plasma levels of HSP10, HSP60 and BiP were measured by immunoassay and related to other plasma measures of inflammation. Periodontal patients had significantly less circulating levels of HSP10 or BiP compared with the controls. In contrast, more periodontal patients had intermediate levels of HSP60. Treatment of the periodontitis caused an increase in plasma levels of HSP10 although it had no effect on BiP. Treatment had no influence of HSP60 levels. Plasma HSP10 levels after therapy correlated with markers of periodontal clin. improvement. Conclusions / Significance. Circulating levels of mol. chaperones are influenced by local inflammation. HSP10 is known to be an anti-inflammatory factor. The marked decrease of this circulating protein in active inflammation and its recovery post-treatment suggests that it may have a role in controlling periodontal inflammation.

RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

AU Shamaei-Tousi, Alireza; D'Aiuto, Francesco; Nibali, Luigi; Steptoe, Andrew; ***Coates, Anthony R. M.*** ; Parkar, Mohamed; Donos, Nikos; Henderson, Brian

IT ***Heat*** - ***shock*** proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(HSP 60; mol. chaperones HSP10, HSP60 and BiP in patients undergoing treatment for periodontal disease)

IT ***Heat*** - ***shock*** proteins
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(HSP10; mol. chaperones HSP10, HSP60 and BiP in patients undergoing treatment for periodontal disease)

L2 ANSWER 3 OF 20 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
DUPLICATE 2

AN 2006:384833 BIOSIS <<LOGINID::20080330>>

DN PREV200600380519

TI Stress wars: the direct role of host and bacterial molecular chaperones in bacterial infection.

AU Henderson, Brian [Reprint Author]; Allan, Elaine; ***CoateS, Anthony R.***
*** M.***

CS Univ Coll London, UCL Eastman Dent Inst, Div Microbial Dis, 256 Grays Inn Rd, London WC1X, UK
b.henderson@eastman.ucl.ac.uk

SO Infection and Immunity, (JUL 2006) Vol. 74, No. 7, pp. 3693-3706.
CODEN: INFIBR. ISSN: 0019-9567.

DT Article
General Review; (Literature Review)

LA English

ED Entered STN: 2 Aug 2006
Last Updated on STN: 2 Aug 2006

AU Henderson, Brian [Reprint Author]; Allan, Elaine; ***CoateS, Anthony R.***
*** M.***

IT . . .
factor-alpha]: expression; chaperonin [EC 3.6.4.9]; peptidyl-prolyl isomerase; geldanamycin: antibacterial-drug, antiinfective-drug; NF-kappa-B; Staphylococcus aureus enterotoxin B: toxin; glyceraldehyde-3-phosphate dehydrogenase [GAPD]; Hsp70 [***heat*** ***shock*** protein70]; Cpn60 [chaperonin 60]; Hsp40/DnaJ; prefoldin nascent chain-associated complex; Hsp32 [heme oxygenase]; DnaK/DnaJ

L2 ANSWER 4 OF 20 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
DUPLICATE 3
AN 2006:506165 BIOSIS <<LOGINID::20080330>>
DN PREV200600501428
TI Association between plasma levels of ***heat*** ***shock***
protein 60 and cardiovascular disease in patients with diabetes mellitus.
AU Shamaei-Tousi, Alireza; Stephens, Jeffrey W.; Bin, Ren; Cooper, Jacqueline
A.; Steptoe, Andrew; ***Coates, Anthony R. M.*** ; Henderson, Brian
[Reprint Author]; Humphries, Steve E.
CS Univ Coll London, Div Microbial Dis, Eastman Dent Inst, 256 Grays Inn Rd,
London WC1X 8LD, UK
b.henderson@eastman.ucl.ac.uk
SO European Heart Journal, (JUL 2006) Vol. 27, No. 13, pp. 1565-1570.
CODEN: EHJODF. ISSN: 0195-668X.
DT Article
LA English
ED Entered STN: 4 Oct 2006
Last Updated on STN: 4 Oct 2006
AB Aims Evidence is accumulating to support the hypothesis that the release
of ***heat*** ***shock*** protein (Hsp)60 into the circulation is
associated with the development of coronary heart disease (CHD). As
diabetes is a risk factor for CHD, it was of interest to determine Hsp60
blood levels in a cross-sectional cohort of diabetic patients, some of
whom had cardiovascular disease, and relate levels to relevant biochemical
markers.Methods and results A total of 855 patients with T1DM or T2DM,
recruited as part of the UCL Diabetes and Cardiovascular disease Study
(UDACS), were assayed for plasma levels of Hsp60. Immunoreactive Hsp60
was detected in 54% of the samples, with 26% having plasma levels > 1 mu
g/mL. Levels of Hsp60 were higher in Caucasians than in other ethnic
groupings, with 56.5% of Caucasian subjects, 37.5% of African-Caribbean
subjects, and 47.1% of Indian subjects having detectable levels (P=0.007),
and with a higher proportion of non-smokers having detectable Hsp60 levels
than smokers (54.9 vs. 43.5%, P=0.01). Of note was the finding of an
association between higher mean plasma levels of Hsp60 in subjects with
clinically manifest cardiovascular disease and those with a history of
myocardial infarction having an adjusted odds ratio of having detectable
Hsp60 of 2.17 (CI 1.26-3.73).Conclusion This is the first report of
circulating Hsp60 levels in diabetic patients, which suggests that this
secreted mitochondrial cell stress protein may be playing an unexpected
role in the cardiovascular pathology associated with diabetes.
TI Association between plasma levels of ***heat*** ***shock***
protein 60 and cardiovascular disease in patients with diabetes mellitus.
AU Shamaei-Tousi, Alireza; Stephens, Jeffrey W.; Bin, Ren; Cooper, Jacqueline
A.; Steptoe, Andrew; ***Coates, Anthony R. M.*** ; Henderson, Brian
[Reprint Author]; Humphries, Steve E.
AB Aims Evidence is accumulating to support the hypothesis that the release
of ***heat*** ***shock*** protein (Hsp)60 into the circulation is
associated with the development of coronary heart disease (CHD). As
diabetes is a risk. . .
IT . . .
endocrine disease/pancreas, metabolic disease
Diabetes Mellitus (MeSH)
IT Diseases
coronary heart disease: heart disease, etiology
Coronary Disease (MeSH)
IT Chemicals & Biochemicals

heat ***shock*** protein 60

L2 ANSWER 5 OF 20 MEDLINE on STN
AN 2006039720 MEDLINE <<LOGINID::20080330>>
DN PubMed ID: 16428728
TI Deletion of the Mycobacterium tuberculosis alpha-crystallin-like hspX gene causes increased bacterial growth in vivo.
AU Hu Yanmin; Movahedzadeh Farahnaz; Stoker Neil G; ***Coates Anthony R*** *** M***
CS Medical Microbiology, Department of Cellular and Molecular Sciences, St. George's Hospital Medical School, London SW17 0RE, United Kingdom.. acoates@sghms.ac.uk
SO Infection and immunity, (2006 Feb) Vol. 74, No. 2, pp. 861-8.
Journal code: 0246127. ISSN: 0019-9567.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 200602
ED Entered STN: 24 Jan 2006
Last Updated on STN: 23 Feb 2006
Entered Medline: 22 Feb 2006
AB Hypervirulent mutants of Mycobacterium tuberculosis, whose growth rates are higher in vivo, have now been reported to have mutations in both regulatory and structural genes, but the basis for this unusual phenotype is not understood. One hypervirulence gene, dosR (devR, Rv2031c), activates transcription of approximately 50 genes in this pathogen in response to hypoxia and nitric oxide stress. The most dramatic activation (approximately 80-fold) is activation of the hspX (acr, Rv2031c) gene, which encodes a 16-kDa alpha-crystallin-like protein that is a major antigen. In this study we found that a Deltaacr mutant exhibited increased growth following infection of BALB/c mice in vivo and in both resting and activated macrophages in vitro (as measured by the number of CFU). The increased growth in macrophages was equal to that of a DeltadosR mutant, while introduction of a constitutively expressed hspX gene reduced the DeltadosR virulence to wild-type levels. These results suggest that the increased number of CFU of the DeltadosR mutant was largely due to loss of hspX expression. We also confirmed that constitutive expression of hspX slows growth in vitro, and we propose that hspX plays an active role in slowing the growth of M. tuberculosis in vivo immediately following infection.
AU Hu Yanmin; Movahedzadeh Farahnaz; Stoker Neil G; ***Coates Anthony R*** *** M***
CT . . .
Proteins: GE, genetics
Bacterial Proteins: ME, metabolism
Cell Line
Cells, Cultured
Colony Count, Microbial
*Gene Deletion
*Gene Expression Regulation, Bacterial
*** Heat-Shock Response***
Humans
Macrophages: MI, microbiology
Mice
Mice, Inbred BALB C

*Mycobacterium tuberculosis: GD, growth & development
Mycobacterium tuberculosis: PY,. . .

L2 ANSWER 6 OF 20 MEDLINE on STN
AN 2005578156 MEDLINE <<LOGINID::20080330>>
DN PubMed ID: 16258145
TI Circulating human ***heat*** ***shock*** protein 60 in the blood of healthy teenagers: a novel determinant of endothelial dysfunction and early vascular injury?.
AU Halcox Julian P J; Deanfield John; Shamaei-Tousi Alireza; Henderson Brian; Steptoe Andrew; ***Coates Anthony R M*** ; Singhal Atul; Lucas Alan
SO Arteriosclerosis, thrombosis, and vascular biology, (2005 Nov) Vol. 25, No. 11, pp. e141-2.
Journal code: 9505803. E-ISSN: 1524-4636.
CY United States
DT Letter
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 200601
ED Entered STN: 1 Nov 2005
Last Updated on STN: 13 Jan 2006
Entered Medline: 12 Jan 2006
TI Circulating human ***heat*** ***shock*** protein 60 in the blood of healthy teenagers: a novel determinant of endothelial dysfunction and early vascular injury?.
AU Halcox Julian P J; Deanfield John; Shamaei-Tousi Alireza; Henderson Brian; Steptoe Andrew; ***Coates Anthony R M*** ; Singhal Atul; Lucas Alan

L2 ANSWER 7 OF 20 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
DUPLICATE 4
AN 2005:244273 BIOSIS <<LOGINID::20080330>>
DN PREV200510030029
TI The intercellular signaling activity of the Mycobacterium tuberculosis chaperonin 60.1 protein resides in the equatorial domain.
AU Tormay, Peter [Reprint Author]; ***Coates, Anthony R. M.*** ; Henderson, Brian
CS St Georges Hosp, Sch Med, Dept Cellular and Mol Med, Cranmer Terrace, London SW17 0RE, UK
p.tormay@sghms.ac.uk
SO Journal of Biological Chemistry, (APR 8 2005) Vol. 280, No. 14, pp. 14272-14277.
CODEN: JBCHA3. ISSN: 0021-9258.
DT Article
LA English
ED Entered STN: 29 Jun 2005
Last Updated on STN: 29 Jun 2005
AB The major ***heat*** ***shock*** protein, chaperonin 60, has been established to have intercellular signaling activity in addition to its established protein-folding function. Mycobacterium tuberculosis is one of a small proportion of bacteria to encode two chaperonin 60 proteins. We have demonstrated that chaperonin 60.1 from this bacterium is a very active stimulator of human monocytes. To determine structure/function relationships of chaperonin 60.1 we have cloned and expressed the apical, equatorial, and intermediate domains of this protein. We have found that the signaling activity of M. tuberculosis chaperonin 60.1 resides in the equatorial domain. This activity of the recombinant equatorial domain was

completely blocked by treating the protein with proteinase K, ruling out lipopolysaccharide contamination as the cause of the cell activation. Blockade of the activity of the equatorial domain by anti-CD14 monoclonal antibodies reveals that this domain activates monocytes by binding to CD14. Looking at the oligomeric state of the active proteins, using native gel electrophoresis and protein cross-linking we found that recombinant M. tuberculosis chaperonin 60.1 fails to form the prototypic tetradecameric structure of chaperonin 60 proteins under the conditions tested and only forms dimers. It is therefore concluded that the monocyte-stimulating activity of M. tuberculosis Cpn60.1 resides in the monomeric subunit and within this subunit the biological activity is due to the equatorial domain.

AU Tormay, Peter [Reprint Author]; ***Coates, Anthony R. M.*** ; Henderson, Brian

AB The major ***heat*** ***shock*** protein, chaperonin 60, has been established to have intercellular signaling activity in addition to its established protein-folding function. Mycobacterium tuberculosis. . .

IT . . .
blood and lymphatics

IT Chemicals & Biochemicals
proteinase K [EC 3.4.21.64]; chaperonin 60.1: equatorial domain, apical domain, intermediate domain, major ***heat*** ***shock*** protein; anti-CD14 monoclonal antibodies [Cpn60.1]

L2 ANSWER 8 OF 20 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN DUPLICATE 5

AN 2006:108759 BIOSIS <<LOGINID::20080330>>

DN PREV200600110304

TI The unusual chaperonins of Mycobacterium tuberculosis.

AU Qamra, Rohini; Mande, Shekhar C. [Reprint Author]; ***Coates, Anthony R.***
*** M.*** ; Henderson, Brian

CS Ctr DNA Fingerprinting and Diagnost, ECIL Rd, Hyderabad 500076, Andhra Pradesh, India
shekhar@cdfd.org.in; a.coates@sghms.ac.uk; b.henderson@eastman.ucl.ac.uk

SO Tuberculosis (Amsterdam), (SEP-NOV 2005) Vol. 85, No. 5-6, pp. 385-394. ISSN: 1472-9792.

DT Article

LA English

ED Entered STN: 8 Feb 2006
Last Updated on STN: 8 Feb 2006

AB ***Heat*** ***shock*** proteins (Hsps), also known as molecular chaperones, are a diverse set of proteins that mediate the correct folding, assembly, transport and degradation of other proteins. In addition, Hsps have been shown to play a variety of important roles in immunity, thereby representing prominent antigens in the humoral and cellular immune response. Chaperonins form a sub-group of molecular chaperones that are found in all domains of life. Chaperonins in all bacteria are encoded by the essential groEL and groES genes, also called cpn60 and cpn10 arranged on the bicistronic groESL operon. Interestingly, Mycobacterium tuberculosis contains two copies of the cpn60 genes. The existence of a duplicate set of cpn60 genes in M. tuberculosis, however, has been perplexing. Cpn10 and Cpn60s of M. tuberculosis have been shown to be highly antigenic in nature, eliciting strong Band T-cell immune responses. Recent work has shown intriguing structural, biochemical and signaling properties of the M. tuberculosis chaperonins. This review details the recent developments in the study of the M. tuberculosis

chaperonins. (c) 2005 Elsevier Ltd. All rights reserved.
AU Qamra, Rohini; Mande, Shekhar C. [Reprint Author]; ***Coates, Anthony R.***
*** M.*** ; Henderson, Brian
AB ***Heat*** ***shock*** proteins (Hsps), also known as molecular chaperones, are a diverse set of proteins that mediate the correct folding, assembly, transport. . .
IT . . .
of Organisms
T cell: immune system, blood and lymphatics; B cell: immune system, blood and lymphatics
IT Chemicals & Biochemicals
heat ***shock*** protein; chaperonin [EC 3.6.4.9]

L2 ANSWER 9 OF 20 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
DUPLICATE 6
AN 2005:305465 BIOSIS <<LOGINID::20080330>>
DN PREV200510089545
TI Comparative cell signalling activity of ultrapure recombinant chaperonin 60 proteins from prokaryotes and eukaryotes.
AU Maguire, Maria; Poole, Stephen; ***Coates, Anthony R. M.*** ; Tormay, Peter; Wheeler-Jones, Caroline; Henderson, Brian [Reprint Author]
CS Univ Coll London, Eastman Dent Inst, Div Microbial Dis, 256 Grays Inn Rd, London WC1X 8LD, UK
maria_maguire2002@yahoo.co.uk
SO Immunology, (JUN 2005) Vol. 115, No. 2, pp. 231-238.
CODEN: IMMUAM. ISSN: 0019-2805.
DT Article
LA English
ED Entered STN: 15 Aug 2005
Last Updated on STN: 15 Aug 2005
AB ***Heat*** - ***shock*** protein (hsp)60/chaperonin 60 is a potent immunogen which has recently been claimed to have cell-signalling actions upon myeloid and vascular endothelial cells. The literature is controversial with different chaperonin 60 proteins producing different patterns of cellular activation and the ever-present criticism that activity is the result of bacterial contaminants. To clarify the situation we have cloned, expressed and purified to homogeneity the chaperonin 60 proteins from Chlamydia pneumoniae, Helicobacter pylori and the human mitochondrion. These highly purified proteins were compared for their ability to stimulate human peripheral blood mononuclear cell (PBMC) cytokine synthesis and vascular endothelial cell adhesion protein expression. In spite of their significant sequence homology, the H. pylori protein was the most potent PBMC activator with the human protein the least potent. PBMC activation by C. pneumoniae and human, but not H. pylori, chaperonin 60 was blocked by antibody neutralization of Toll-like receptor-4. The C. pneumoniae chaperonin 60 was the most potent endothelial cell activator, with the human protein being significantly less active than bacterial chaperonin 60 proteins. These results have implications for the role of chaperonin 60 proteins as pathological factors in autoimmune and cardiovascular disease, and raise the possibility that each of these proteins may result in different pathological effects in such diseases.
AU Maguire, Maria; Poole, Stephen; ***Coates, Anthony R. M.*** ; Tormay, Peter; Wheeler-Jones, Caroline; Henderson, Brian [Reprint Author]
AB ***Heat*** - ***shock*** protein (hsp)60/chaperonin 60 is a potent immunogen which has recently been claimed to have cell-signalling actions

upon myeloid and vascular. . .
IT . . .
& Systems of Organisms
peripheral blood mononuclear cell: immune system, blood and lymphatics; mitochondrion
IT Chemicals & Biochemicals
chaperonin 60 [***heat*** ***shock*** protein-60]: expression

L2 ANSWER 10 OF 20 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2006:36438 CAPLUS <<LOGINID::20080330>>
DN 144:409874
TI Circulating human ***heat*** ***shock*** protein 60 in the blood of healthy teenagers: A novel determinant of endothelial dysfunction and early vascular injury?
AU Halcox, Julian P. J.; Deanfield, John; Shamaei-Tousi, Alireza; Henderson, Brian; Steptoe, Andrew; ***Coates, Anthony R. M.*** ; Singhal, Atul; Lucas, Alan
CS Vascular Physiology Unit, Department of Cardiology, Institute of Child Health, University College London, UK
SO Stroke (2005), 36(11), e141-e142
CODEN: SJCCA7; ISSN: 0039-2499
PB Lippincott Williams & Wilkins
DT Journal
LA English
AB The potential role for ***heat*** - ***shock*** protein 60 (HSP60) in early atherogenesis was studied in 294 healthy adolescents aged 13-16 years. Serum from 256 subjects was available for anal. Sol. HSP60 (sHSP60) was detectable in 60 subjects over a wide range of values. Results showed that teenagers with detectable sHSP60 had significantly lower flow-mediated vasodilatation (FMD) than the remainder who lacked this stress protein in their blood. A subtle relationship between loge[sHSP60] levels and FMD was obsd. HDL cholesterol levels were slightly higher, but other variableswere similar in subjects with and without detectable sHSP60. These findings suggest that sHSP60 or factors that stimulate the expression and systemic release of HSP60, may contribute to the initiation of arterial disease in early life.
RE.CNT 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
TI Circulating human ***heat*** ***shock*** protein 60 in the blood of healthy teenagers: A novel determinant of endothelial dysfunction and early vascular injury?
AU Halcox, Julian P. J.; Deanfield, John; Shamaei-Tousi, Alireza; Henderson, Brian; Steptoe, Andrew; ***Coates, Anthony R. M.*** ; Singhal, Atul; Lucas, Alan
AB The potential role for ***heat*** - ***shock*** protein 60 (HSP60) in early atherogenesis was studied in 294 healthy adolescents aged 13-16 years. Serum from 256 subjects was. . .
ST ***heat*** ***shock*** protein 60 marker endothelial dysfunction adolescent
IT ***Heat*** - ***shock*** proteins
RL: BSU (Biological study, unclassified); DGN (Diagnostic use); BIOL (Biological study); USES (Uses)
(HSP 60; circulating HSP60 in blood of healthy teenagers as novel determinant of endothelial dysfunction and early vascular injury)

L2 ANSWER 11 OF 20 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2005:1250549 CAPLUS <<LOGINID::20080330>>

TI Circulating human ***heat*** ***shock*** protein 60 in the blood of healthy teenagers: A novel determinat of endothelial dysfunction and early vascular injury?
AU Halcox, Julian P. J.; Deanfield, John; Shamaei-Tousi, Alireza; Henderson, Brian; Steptoe, Andrew; ***Coates, Anthony R. M.*** ; Singhal, Atul; Lucas, Alan
CS Vascular Physiology Unit, Department of Cardiology, Institute of Child Health, University College London, UK
SO Arteriosclerosis, Thrombosis, and Vascular Biology (2005), 25(11), e141-e142
CODEN: ATVBFA; ISSN: 1079-5642
PB Lippincott Williams & Wilkins
DT Journal; Letter
LA English
AB Unavailable
RE.CNT 11 THERE ARE 11 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
TI Circulating human ***heat*** ***shock*** protein 60 in the blood of healthy teenagers: A novel determinat of endothelial dysfunction and early vascular injury?
AU Halcox, Julian P. J.; Deanfield, John; Shamaei-Tousi, Alireza; Henderson, Brian; Steptoe, Andrew; ***Coates, Anthony R. M.*** ; Singhal, Atul; Lucas, Alan

L2 ANSWER 12 OF 20 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2004:119531 CAPLUS <<LOGINID::20080330>>
DN 140:175161
TI ***Heat*** ***shock*** polypeptides as pain relief agents
IN ***Coates, Anthony Robert Milnes***
PA Helperby Therapeutics Limited, UK
SO Brit. UK Pat. Appl., 39 pp.
CODEN: BAXXDU
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI GB 2391477	A	20040211	GB 2003-25782	20031105
GB 2391477	B	20041222		
CA 2503964	A1	20040521	CA 2003-2503964	20031105
WO 2004041304	A2	20040521	WO 2003-GB4774	20031105
WO 2004041304	A3	20040729		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003279454	A1	20040607	AU 2003-279454	20031105
EP 1562625	A2	20050817	EP 2003-772402	20031105
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
BR 2003016053	A	20050920	BR 2003-16053	20031105

	CN 1735427	A	20060215	CN 2003-80108562	20031105
	JP 2006514001	T	20060427	JP 2004-549345	20031105
	NO 2005002216	A	20050805	NO 2005-2216	20050509
	US 2006252681	A1	20061109	US 2006-534054	20060322
PRAI	GB 2002-26105	A	20021108		
	WO 2003-GB4774	W	20031105		

AB The present invention concerns the use of a ***heat*** ***shock*** polypeptide and/or an encoding nucleic acid sequence in the manuf. of a medicament for use in the relief of pain. In particular the invention concerns the use of chaperonin. The invention further provides methods of relieving pain using medicaments contg. the ***heat*** ***shock*** polypeptides.

RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI ***Heat*** ***shock*** polypeptides as pain relief agents

IN ***Coates, Anthony Robert Milnes***

AB The present invention concerns the use of a ***heat*** ***shock*** polypeptide and/or an encoding nucleic acid sequence in the manuf. of a medicament for use in the relief of pain.. . . particular the invention concerns the use of chaperonin. The invention further provides methods of relieving pain using medicaments contg. the ***heat*** ***shock*** polypeptides.

ST pain relief ***heat*** ***shock*** polypeptides nucleic acid; chaperonin pain relief nucleic acid

IT Disease, animal
(back pain; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Body, anatomical
(back, disease, pain; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Pain
(back; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Drug delivery systems
(carriers; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Pain
(dental; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Drug delivery systems
(diluents; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Viscera
(disease, pain; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Ear, disease
(earache; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Drug delivery systems

(excipients; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Bone, disease
(fracture, pain from; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Nucleic acids
RL: BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(***heat*** ***shock*** protein-encoding; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Eubacteria
Mycobacterium
Mycobacterium tuberculosis
(***heat*** ***shock*** proteins from; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Analgesics
Animals
Drug interactions
Headache
Human
Pain
(***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT ***Heat*** - ***shock*** proteins
Molecular chaperones
Opioids
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Drug delivery systems
(inhalants; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Drug delivery systems
(nasal; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Anti-inflammatory agents
(nonsteroidal; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Drug delivery systems
(ophthalmic; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Drug delivery systems
(oral; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Abscess

Arthritis
Burn
Gout
Infection
Inflammation
Menstruation
Neoplasm
Parturition
(pain from; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Tooth, disease
(pain; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Drug delivery systems
(parenterals; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Surgery
(post-surgical pain from; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Kidney, disease
(renal tract pain; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Animal tissue, disease
(soft, injury, pain from; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Drug delivery systems
(suppositories, vaginal; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Drug delivery systems
(suppositories; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Drug delivery systems
(topical; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Ligament
Tendon
(traumatic damage, pain from; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Disease, animal
(visceral pain; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Pain
(visceral; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT 657078-79-0, Chaperonin 60.2 (Mycobacterium tuberculosis) 657078-80-3,

Chaperonin 10 (Mycobacterium tuberculosis)
RL: BSU (Biological study, unclassified); PAC (Pharmacological activity); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(amino acid sequence; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)
IT 657085-31-9
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(amino acid sequence; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)
IT 50-78-2, Aspirin 103-90-2, Paracetamol 15687-27-1, Ibuprofen
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)
IT 329900-75-6, Cyclooxygenase 2
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)
IT 657078-76-7 657078-77-8 657078-78-9
RL: BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(nucleotide sequence; ***heat*** ***shock*** proteins that are chaperonins as pain relief agents and their encoding nucleic acids in relation to use with other analgesics)

L2 ANSWER 13 OF 20 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN DUPLICATE 7
AN 2001:18271 BIOSIS <<LOGINID::20080330>>
DN PREV200100018271
TI Detection of mRNA transcripts and active transcription in persistent Mycobacterium tuberculosis induced by exposure to rifampin or pyrazinamide.
AU Hu, Yanmin; Mangan, Joseph A.; Dhillon, Jasvir; Sole, Kath M.; Mitchison, Denis A.; Butcher, Philip D. [Reprint author]; ***Coates, Anthony R.*** ***M.***
CS Department of Medical Microbiology, St. George's Hospital Medical School, Cranmer Terrace, London, SW17 0RE, UK
butcherp@sghms.ac.uk
SO Journal of Bacteriology, (November, 2000) Vol. 182, No. 22, pp. 6358-6365. print.
CODEN: JOBAAY. ISSN: 0021-9193.
DT Article
LA English
ED Entered STN: 27 Dec 2000
Last Updated on STN: 27 Dec 2000
AB Mycobacterium tuberculosis can persist in an altered physiological state for many years after initial infection, and it may reactive to cause active disease. An analogous persistent state, possibly consisting of several different subpopulations of bacteria, may arise during chemotherapy; this state is thought to be responsible for the prolonged period required for effective chemotherapy. Using two models of

drug-induced persistence, we show that both microaerophilic stationary-phase M. tuberculosis treated with a high dose of rifampin in vitro and pyrazinamide-induced persistent bacteria in mice are nonculturable yet still contain 16S rRNA and mRNA transcripts. Also, the in vitro persistent, plate culture-negative bacteria incorporate radioactive uridine into their RNA in the presence of rifampin and can rapidly up-regulate gene transcription after the replacement of the drug with fresh medium and in response to ***heat*** ***shock*** . Our results show that persistent M. tuberculosis has transcriptional activity. This finding provides a molecular basis for the rational design of drugs targeted at persistent bacteria.

AU Hu, Yanmin; Mangan, Joseph A.; Dhillon, Jasvir; Sole, Kath M.; Mitchison, Denis A.; Butcher, Philip D. [Reprint author]; ***Coates, Anthony R.*** *** M.***

AB. . . rifampin and can rapidly up-regulate gene transcription after the replacement of the drug with fresh medium and in response to ***heat*** ***shock*** . Our results show that persistent M. tuberculosis has transcriptional activity. This finding provides a molecular basis for the rational design. . .

IT Miscellaneous Descriptors
drug-induced persistence; gene transcription up-regulation; ***heat*** ***shock*** ; microaerophilic stationary-phase

L2 ANSWER 14 OF 20 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AN 1999:442588 BIOSIS <<LOGINID::20080330>>

DN PREV199900442588

TI The unfolding story of the chaperonins.

AU ***Coates, Anthony R.M.*** [Reprint author]; Henderson, Brian; Mascagni, Paolo

CS Department of Medical Microbiology, St George's Hospital Medical School, Cranmer Terrace, London, SW17 0RE, UK

SO Harding, S. E. [Editor]. Biotechnol. Genet. Eng. Rev., (1999) pp. 393-405. Biotechnology and Genetic Engineering Reviews. print.
Publisher: Intercept Ltd., P. O. Box 716, Andover SP10 1YG, England.
Series: Biotechnology and Genetic Engineering Reviews.
CODEN: BGERES. ISSN: 0264-8725. ISBN: 1-898298-58-0.

DT Book
Book; (Book Chapter)

LA English

ED Entered STN: 26 Oct 1999
Last Updated on STN: 26 Oct 1999

AU ***Coates, Anthony R.M.*** [Reprint author]; Henderson, Brian; Mascagni, Paolo

IT . . .
Biochemicals
chaperonin 10: cellular location; chaperonin 60: cellular location; chaperonin: cellular location, pharmaceutical, industrial protein; early pregnancy factor: cellular location; ***heat*** ***shock*** protein 10: cellular location; ***heat*** ***shock*** protein 58/60/65: cellular location; protein: folding, structure; P1: cellular location; chaperonin gene: organization

L2 ANSWER 15 OF 20 CAPLUS COPYRIGHT 2008 ACS on STN

AN 1999:488005 CAPLUS <<LOGINID::20080330>>

DN 131:282725

TI The unfolding story of the chaperonins

AU ***Coates, Anthony R. M.*** ; Henderson, Brian; Mascagni, Paolo
CS Department of Medical Microbiology, St George's Hospital Medical School, London, SW17 0RE, UK
SO Biotechnology & Genetic Engineering Reviews (1999), 16, 393-405
CODEN: BGERES; ISSN: 0264-8725
PB Intercept Ltd.
DT Journal; General Review
LA English
AB A review, with .apprx.65 refs., on the recent advances in the folding functions of GroE subfamily which contains GroES, GroEL, and hsp10.
RE.CNT 80 THERE ARE 80 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
AU ***Coates, Anthony R. M.*** ; Henderson, Brian; Mascagni, Paolo
IT ***Heat*** - ***shock*** proteins
RL: PEP (Physical, engineering or chemical process); PRP (Properties); PROC (Process)
(10; protein folding functions of chaperonins)

L2 ANSWER 16 OF 20 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN DUPLICATE 8
AN 1997:516395 BIOSIS <<LOGINID::20080330>>
DN PREV199799815598
TI Mycobacterium tuberculosis chaperonin 10 stimulates bone resorption: A potential contributory factor in Pott's disease.
AU Meghji, Sajeda; White, Peter A.; Nair, Sean P.; Reddi, Krisanavane; Heron, Kyle; Henderson, Brian; Zaliani, Andrea; Fossati, Gianluca; Mascagni, Paolo; Hunt, John F.; Roberts, Michael M. [Reprint author]; ***Coates,*** *** Anthony R. M.***
CS Room 1.241A, Jenner Wing, Level 1, Div. Mol. Microbiol., St. George's Hosp. Med. Sch., Cranmer Terrace, London SW17 ORE, UK
SO Journal of Experimental Medicine, (1997) Vol. 186, No. 8, pp. 1241-1246.
CODEN: JEMEAV. ISSN: 0022-1007.
DT Article
LA English
ED Entered STN: 10 Dec 1997
Last Updated on STN: 10 Dec 1997
AB Pott's disease (spinal tuberculosis), a condition characterized by massive resorption of the spinal vertebrae, is one of the most striking pathologies resulting from local infection with Mycobacterium tuberculosis (Mt; Boachie-Adjei, O., and R,.G. Squillante. 1996. Orthop. Clin. North Am. 27:95-103). The pathogenesis of Pott's disease is not established. Here we report for the first time that a protein, identified by a monoclonal antibody to be the Mt ***heat*** ***shock*** protein (Baird, P.N., L.M. Hall, and A.R.M. Coates. 1989. J. Gen. Microbiol. 135:931-939) chaperonin (cpn) 10, is responsible for the osteolytic activity of this bacterium. Recombinant Mt cpn10 is a potent stimulator of bone resorption in bone explant cultures and induces osteoclast recruitment, while inhibiting the proliferation of an osteoblast bone-forming cell line. Furthermore, we have found that synthetic peptides corresponding to sequences within the flexible loop and sequence 65-70 of Mt cpn10 may comprise a single conformational unit which encompasses its potent bone-resorbing activity. Our findings suggest that Mt cpn10 may be a valuable pharmacological target for the clinical therapy of vertebral tuberculosis and possibly other bone diseases.
AU. . . Reddi, Krisanavane; Heron, Kyle; Henderson, Brian; Zaliani, Andrea; Fossati, Gianluca; Mascagni, Paolo; Hunt, John F.; Roberts, Michael M. [Reprint author]; ***Coates, Anthony R. M.***

AB. . . established. Here we report for the first time that a protein, identified by a monoclonal antibody to be the Mt ***heat*** ***shock*** protein (Baird, P.N., L.M. Hall, and A.R.M. Coates. 1989. J. Gen. Microbiol. 135:931-939) chaperonin (cpn) 10, is responsible for the. . .

L2 ANSWER 17 OF 20 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 1995:346931 BIOSIS <<LOGINID::20080330>>
DN PREV199598361231
TI Sequence and structural homologies between M. tuberculosis chaperonin 10 the MHC class I/II peptide binding cleft.
AU Chan, Edith; Fossati, Gianluca; Giuliani, Paola; Lucietto, Pierluigi; Zaliani, Andrea; ***Coates, Antony R. M.*** ; Mascagni, Paolo [Reprint author]
CS Italfarmaco Res. Centre, Via Lavoratori 54, Cinisello B, 20092 Milan, Italy
SO Biochemical and Biophysical Research Communications, (1995) Vol. 211, No. 1, pp. 14-20.
CODEN: BBRCA9. ISSN: 0006-291X.
DT Article
LA English
ED Entered STN: 10 Aug 1995
Last Updated on STN: 10 Aug 1995
AB The peptide corresponding to the C-terminal half of M. tuberculosis hsp10 was synthesised based on the prediction that it might represent an independent structural region of the protein. This hypothesis was confirmed by aggregation and CD studies using this peptide and longer sequences of the protein. The peptide shares about 40-50% sequence homology with alpha-2 and beta-1 chains of MHC class I and II antigens. This and the CD results which indicated that the peptide at acidic pHs folds into an anti-parallel beta-sheet were used to generate a 3D model which has the same "W" fold contained in the MHC peptide binding groove. These data suggest that the hypothesis of molecular mimicry proposed to be one of the mechanisms which triggers autoimmune diseases may be extended to hsp10 proteins. Furthermore the suggested evolutionary relationship between hsp's and MHC antigens may find support from these data.
AU Chan, Edith; Fossati, Gianluca; Giuliani, Paola; Lucietto, Pierluigi; Zaliani, Andrea; ***Coates, Antony R. M.*** ; Mascagni, Paolo [Reprint author]
IT Miscellaneous Descriptors
AUTOIMMUNE DISEASE; ***HEAT*** ***SHOCK*** PROTEIN; MAJOR HISTOCOMPATIBILITY COMPLEX; PROTEIN SYNTHESIS

L2 ANSWER 18 OF 20 CAPLUS COPYRIGHT 2008 ACS on STN
AN 1989:588440 CAPLUS <<LOGINID::20080330>>
DN 111:188440
TI Identification of two unknown reading frames in Synechococcus 6301 as homologs of the 10k and 65k antigen genes of Mycobacterium tuberculosis and related ***heat*** ***shock*** genes in E. coli and Coxiella burnetii
AU Cookson, M. John; Baird, Paul N.; Hall, Lucinda M. C.; ***Coates,*** *** Anthony R. M.***
CS Med. Coll., London Hosp., London, E1 2AD, UK
SO Nucleic Acids Research (1989), 17(15), 6392
CODEN: NARHAD; ISSN: 0305-1048
DT Journal

LA English
AB The translation products of 2 unknown reading frames reported from the cyanobacterium Synechococcus 6301 have sequence homol. with the 10k and 65k ***heat*** ***shock*** proteins of M. tuberculosis, E. coli, and C. burnetii. The unknown reading frames are URF-4 and URF-3, which are adjacent to an ATP synthase locus.
TI . . . unknown reading frames in Synechococcus 6301 as homologs of the 10k and 65k antigen genes of Mycobacterium tuberculosis and related ***heat*** ***shock*** genes in E. coli and Coxiella burnetii
AU Cookson, M. John; Baird, Paul N.; Hall, Lucinda M. C.; ***Coates,*** ***Anthony R. M.***
AB . . . products of 2 unknown reading frames reported from the cyanobacterium Synechococcus 6301 have sequence homol. with the 10k and 65k ***heat*** ***shock*** proteins of M. tuberculosis, E. coli, and C. burnetii. The unknown reading frames are URF-4 and URF-3, which are adjacent. . .
ST Synechococcus ***heat*** ***shock*** protein sequence
IT Gene and Genetic element, microbial
RL: BIOL (Biological study)
(for ***heat*** ***shock*** -related proteins, of Synechococcus 6301)
IT Coxiella burnetii
Escherichia coli
Mycobacterium tuberculosis
(***heat*** ***shock*** proteins of, Synechococcus 6301 homologs of)
IT Synechococcus
(***heat*** ***shock*** -related proteins of, structure of)
IT Proteins, specific or class
RL: BIOL (Biological study)
(***heat*** - ***shock*** -related, URF 3, of Synechococcus 6301, structure of)
IT Proteins, specific or class
RL: BIOL (Biological study)
(***heat*** - ***shock*** -related, URF 4, of Synechococcus 6301, structure of)

L2 ANSWER 19 OF 20 CAPLUS COPYRIGHT 2008 ACS on STN
AN 1990:71041 CAPLUS <<LOGINID::20080330>>
DN 112:71041
TI Cloning and sequence analysis of the 10 kDa antigen gene of Mycobacterium tuberculosis
AU Baird, Paul N.; Hall, Lucinda M. C.; ***Coates, Anthony R. M.***
CS Med. Coll., London Hosp., London, E1 2AD, UK
SO Journal of General Microbiology (1989), 135(4), 931-9
CODEN: JGMIAN; ISSN: 0022-1287
DT Journal
LA English
AB The gene encoding a major protein antigen of M. tuberculosis was cloned and sequenced using oligonucleotide probes derived from the N-terminal sequence of the analogous protein from M. bovis BCG. The gene anal. revealed a sequence encoding a protein of 99 amino acid residues, with a mol. mass of 10.7 kDa. Computer prediction suggests that the protein contains three T-cell-detd. epitopes (of which one has been demonstrated exptl.) and three B-cell-detd. epitopes. The protein sequence was homologous to two prokaryote ***heat*** - ***shock*** proteins, and the gene possessed ***heat*** - ***shock*** -like promoter sequences

upstream of the initiation codon. A hairpin loop identified in the upstream sequence may also be important in regulation of the gene.
AU Baird, Paul N.; Hall, Lucinda M. C.; ***Coates, Anthony R. M.***
AB . . . epitopes (of which one has been demonstrated exptl.) and three B-cell-detd. epitopes. The protein sequence was homologous to two prokaryote ***heat*** - ***shock*** proteins, and the gene possessed ***heat*** - ***shock*** -like promoter sequences upstream of the initiation codon. A hairpin loop identified in the upstream sequence may also be important in. . .

L2 ANSWER 20 OF 20 CAPLUS COPYRIGHT 2008 ACS on STN
AN 1989:1814 CAPLUS <<LOGINID::20080330>>
DN 110:1814
TI A major antigen from Mycobacterium tuberculosis which is homologous to the ***heat*** ***shock*** proteins groES from E.coli and the htpA gene product of Coxiella burneti
AU Baird, Paul N.; Hall, Lucinda M. C.; ***Coates, Anthony R. M.***
CS Med. Coll., London Hosp., London, E1 2AD, UK
SO Nucleic Acids Research (1988), 16(18), 9047
CODEN: NARHAD; ISSN: 0305-1048
DT Journal
LA English
AB A 10-kilodalton (kDa) protein antigen of M. tuberculosis is recognized by sera from tuberculosis patients and its sequence was detd. Homol. was demonstrated between the 10-kDa antigen of M. tuberculosis and the ***heat*** - ***shock*** proteins encoded by Escherichia coli gene groES and C. burneti gene htpA. The M. tuberculosis 10-kDa protein comprises 99 amino acids and is 44% homologous with the groES gene prodn. and 43% homologous with the htpA gene product. This suggests that the 10-kDa protein of M. tuberculosis may have as wide a distribution among organisms as the 65-kDa antigen which is also homologous to 2 other major ***heat*** - ***shock*** proteins of E. coli and C. burneti.
TI A major antigen from Mycobacterium tuberculosis which is homologous to the ***heat*** ***shock*** proteins groES from E.coli and the htpA gene product of Coxiella burneti
AU Baird, Paul N.; Hall, Lucinda M. C.; ***Coates, Anthony R. M.***
AB . . . from tuberculosis patients and its sequence was detd. Homol. was demonstrated between the 10-kDa antigen of M. tuberculosis and the ***heat*** - ***shock*** proteins encoded by Escherichia coli gene groES and C. burneti gene htpA. The M. tuberculosis 10-kDa protein comprises 99 amino. . . may have as wide a distribution among organisms as the 65-kDa antigen which is also homologous to 2 other major ***heat*** - ***shock*** proteins of E. coli and C. burneti.
ST Mycobacterium antigen homol ***heat*** ***shock*** protein; sequence antigen Mycobacterium; Escherichia ***heat*** ***shock*** protein homol Mycobacterium; Coxiella ***heat*** ***shock*** protein homol Mycobacterium; gene groES Escherichia ***heat*** ***shock*** protein
IT Mycobacterium tuberculosis
(10 kDa protein antigen of, sequence of and Escherichia coli gene groES and Coxiella burneti gene htpA ***heat*** - ***shock*** proteins homol. to)
IT Escherichia coli
(***heat*** - ***shock*** protein encoded by gene groES of, Mycobacterium tuberculosis 10 kDa protein antigen homol. to)
IT Coxiella burnetii
(***heat*** - ***shock*** protein encoded by gene htpA of,

```
        Mycobacterium tuberculosis 10 kDa protein antigen homol. to)
IT   Antigens
        (of Mycobacterium tuberculosis, Escherichia coli gene groES and
        Coxiella burneti gene htpA   ***heat***  -  ***shock***   proteins
        homol. to and amino acid sequence of)
IT   Protein sequences
        (of gene groES   ***heat***  -  ***shock***   protein, of Escherichia
        coli, complete)
IT   Protein sequences
        (of gene htpA   ***heat***  -  ***shock***   protein, of Coxiella
        burneti, complete)
IT   Proteins, specific or class
        (gene groES,   ***heat***  -  ***shock***  , of Escherichia coli,
        Mycobacterium tuberculosis 10 kDa protein antigen homol. to)
IT   Proteins, specific or class
        (gene htpA,   ***heat***  -  ***shock***  , of Coxiella burneti,
        Mycobacterium tuberculosis 10 kDa protein antigen homol. to)
IT   Gene and Genetic element, microbial
        (groES,   ***heat***  -  ***shock***   protein encoded by, of
        Escherichia coli, Mycobacterium tuberculosis 10 kDa protein antigen
        homol. to)
IT   Gene and Genetic element, microbial
        (htpA,   ***heat***  -  ***shock***   protein encoded by, of Coxiella
        burneti, Mycobacterium tuberculosis 10 kDa protein antigen homol. to)


=> s pain and ((heat shock protein?)or(chaparonin?))
L3         130 PAIN AND ((HEAT SHOCK PROTEIN?) OR(CHAPARONIN?))

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4           92 DUP REM L3 (38 DUPLICATES REMOVED)

=> s l4 and (pain/ti or pain/ab)
'AB' IS NOT A VALID FIELD CODE
'AB' IS NOT A VALID FIELD CODE
L5          65 L4 AND (PAIN/TI OR PAIN/AB)

=> s l5 and (shock/ti or shock/ab)
'AB' IS NOT A VALID FIELD CODE
'AB' IS NOT A VALID FIELD CODE
L6          41 L5 AND (SHOCK/TI OR SHOCK/AB)

=> s l6 and (chaparon?/ti or chaparon?/ab)
'AB' IS NOT A VALID FIELD CODE
'AB' IS NOT A VALID FIELD CODE
L7           0 L6 AND (CHAPARON?/TI OR CHAPARON?/AB)

=> d bib ab kwic l6 1-
YOU HAVE REQUESTED DATA FROM 41 ANSWERS - CONTINUE? Y/(N):y

L6   ANSWER 1 OF 41  BIOSIS  COPYRIGHT (c) 2008 The Thomson Corporation  on STN
AN   2007:391673  BIOSIS <<LOGINID::20080330>>
DN   PREV200700396079
TI   Cyclooxygenase (COX) inhibitors and the intestine.
AU   Little, Dianne; Jones, Samuel L.; Blikslager, Anthony T. [Reprint Author]
CS   N Carolina State Univ, Coll Vet Med, Dept Clin Sci, Col and Digest Dis
```

Program, 4700 Hillsborough St, Raleigh, NC 27606 USA
Anthony.Blikslager@ncsu.edu
SO Journal of Veterinary Internal Medicine, (MAY-JUN 2007) Vol. 21, No. 3, pp. 367-377.
ISSN: 0891-6640.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 18 Jul 2007
Last Updated on STN: 18 Jul 2007
AB Nonsteroidal anti-inflammatory drugs (NSAIDs) have long been used for the treatment of ***pain*** and inflammation because of their inhibitory effects on cyclooxygenase (COX). For almost as long as NSAIDs have been in use, multiple adverse effects have been noted. Assessment of many of these adverse effects have been complicated because of the discovery of multiple splice variants of the cox gene, and a greater array of COX inhibitors, especially the COX-2 selective inhibitors have become available. Some of these adverse effects cannot be readily explained by the effect of these drugs on COX. This has sparked a new field of investigation into the COX-independent effects of the COX inhibitors. The major noncyclooxygenase targets of the COX inhibitors of particular relevance to inflammation and the gastrointestinal tract are phosphatidylinositol 3'-kinase Akt signaling, uncoupling of oxidative phosphorylation, PPAR gamma, nuclear factor kappa B, mitogen activated protein kinases, and ***heat*** ***shock*** ***proteins*** .
AB Nonsteroidal anti-inflammatory drugs (NSAIDs) have long been used for the treatment of ***pain*** and inflammation because of their inhibitory effects on cyclooxygenase (COX). For almost as long as NSAIDs have been in use,. . . are phosphatidylinositol 3'-kinase Akt signaling, uncoupling of oxidative phosphorylation, PPAR gamma, nuclear factor kappa B, mitogen activated protein kinases, and ***heat*** ***shock*** ***proteins*** .
IT . . .
immune system disease, drug therapy
IT Chemicals & Biochemicals
Akt; mitogen activated protein kinase [EC 2.7.1.37]; phosphatidylinositol 3'-kinase [EC 2.7.1.137]; ***heat*** ***shock*** ***protein*** ; nuclear factor kappa B; PPAR gamma; cyclooxygenase [COX] [EC 1.14.99.1]; nonsteroidal anti-inflammatory drugs [NSAIDs]: enzyme inhibitor-drug, immunologic-drug, antiinflammatory-drug

L6 ANSWER 2 OF 41 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2007:313987 BIOSIS <<LOGINID::20080330>>
DN PREV200700311460
TI A pilot study with a therapeutic vaccine based on hydroxyapatite ceramic particles and selfmantigens in cancer patients.
AU Ciocca, Daniel R. [Reprint Author]; Frayssinet, Patrick; Cuello-Carrion, F. Dario
CS Argentine Fdn Canc Res, Sargento Cabral 706, RA-5500 Mendoza, Argentina
dciocca@lab.cricyt.edu.ar
SO Cell Stress & Chaperones, (SPR 2007) Vol. 12, No. 1, pp. 33-43.
ISSN: 1355-8145.
DT Article
LA English
ED Entered STN: 16 May 2007
Last Updated on STN: 16 May 2007

AB We describe an approach to produce an autologous therapeutic antitumor vaccine using hydroxyapatite (HA) for vaccinating cancer patients. The novel approach involved (1) the purification of part of the self-tumor antigens/ adjuvants using column chromatography with HA, (2) the employ of HA as a medium to attract antigen-presenting cells (APCs) to the vaccination site, and (3) the use of HA as a vector to present in vivo the tumor antigens and adjuvants to the patient's APCs. The vaccine was prepared using and combining HA particles, with at least 3 ***heat*** ***shock*** ***proteins*** (gp96 was one of them possibly with chaperoned proteins/peptides as shown in the slot blots) and with proteins from the cell membrane system (including Hsp70, Hsp27, and membrane proteins). The timing of HA degradation was tested in rats; the HA particles administered under the skin attracted macrophages and were degraded into smaller particles, and they were totally phagocytized within 1 week. In patients (n = 20), the vaccine was then administered weekly and showed very low toxicity, causing minor and tolerable local inflammation (erythema, papule, or local ***pain***); only 1 patient who received a larger dose presented hot flashes, and there were no systemic manifestations of toxicity or autoimmune diseases attributed to the vaccine. Our study suggests that this therapeutic vaccine has shown some efficacy producing a positive response in certain patients. Stable disease was noted in 25% of the patients (renal carcinoma, breast carcinoma, and astrocytoma), and a partial response was noted in 15% of the patients (breast carcinoma and astrocytoma). The most encouraging results were seen in patients with recurrent disease; 4 patients in these conditions (20%) are disease free following the vaccine administration. However, we do not want to overstate the clinical efficacy in this small number of patients. The therapeutic vaccine tested in our study is working by activating the T-cell response as was shown in the comparative histological and immunohistochemical study performed in the pre- and postvaccine biopsy taken from a patient with inflammatory breast carcinoma. However, we cannot ruled out that the vaccine could also be producing an antibody(ies)-mediated response. In conclusion, this therapeutic vaccine based on HA ceramic particles and self-antigens can be safely administered and is showing some encouraging clinical results in cancer patients.

AB. . . antigens and adjuvants to the patient's APCs. The vaccine was prepared using and combining HA particles, with at least 3 ***heat*** ***shock*** ***proteins*** (gp96 was one of them possibly with chaperoned proteins/peptides as shown in the slot blots) and with proteins from the. . . vaccine was then administered weekly and showed very low toxicity, causing minor and tolerable local inflammation (erythema, papule, or local ***pain***); only 1 patient who received a larger dose presented hot flashes, and there were no systemic manifestations of toxicity or. . .

IT . . .
neoplastic disease
Astrocytoma (MeSH)

IT Diseases
renal carcinoma: urologic disease, neoplastic disease
Kidney Neoplasms (MeSH); Carcinoma (MeSH)

IT Chemicals & Biochemicals
hydroxyapatite; ***heat*** ***shock*** ***protein*** 70; membrane protein; antigen; ***heat*** ***shock*** ***protein*** 27; vaccine

L6 ANSWER 3 OF 41 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AN 2007:213912 BIOSIS <<LOGINID::20080330>>
DN PREV200700217387
TI Thiazolidinedione class of peroxisome proliferator-activated receptor gamma agonists prevents neuronal damage, motor dysfunction, myelin loss, neuropathic ***pain*** , and inflammation after spinal cord injury in adult rats.
AU Park, Seung-Won; Yi, Jae-Hyuk; Miranpuri, Guruwattan; Satriotomo, Irawan; Bowen, Kellie; Resnick, Daniel K.; Vemuganti, Raghu [Reprint Author]
CS Univ Wisconsin, Dept Neurosurg, Neurosci Training Program, K4-8,Mail Stop Code CSC-8660,600 Highland Ave, Madison, WI 53792 USA vemugant@neurosurg.wisc.edu
SO Journal of Pharmacology and Experimental Therapeutics, (MAR 2007) Vol. 320, No. 3, pp. 1002-1012. http://www.jpet.org.
CODEN: JPETAB. ISSN: 0022-3565.
DT Article
LA English
ED Entered STN: 28 Mar 2007
Last Updated on STN: 28 Mar 2007
AB Thiazolidinediones (TZDs) are potent synthetic agonists of the ligand-activated transcription factor peroxisome proliferator-activated receptor-gamma (PPAR gamma). TZDs were shown to induce neuroprotection after cerebral ischemia by blocking inflammation. As spinal cord injury (SCI) induces massive inflammation that precipitates secondary neuronal death, we currently analyzed the therapeutic efficacy of TZDs pioglitazone and rosiglitazone after SCI in adult rats. Both pioglitazone and rosiglitazone (1.5 mg/kg i.p.; four doses at 5 min and 12, 24, and 48 h) significantly decreased the lesion size (by 57 to 68%, $p < 0.05$), motor neuron loss (by 3- to 10-fold, $p < 0.05$), myelin loss (by 66 to 75%, $p < 0.05$), astrogliosis (by 46 to 61%, $p < 0.05$), and microglial activation (by 59 to 78%, $p < 0.05$) after SCI. TZDs significantly enhanced the motor function recovery (at 7 days after SCI, the motor scores were 37 to 45% higher in the TZD groups over the vehicle group; $p < 0.05$), but the treatment was effective only when the first injection was given by 2 h after SCI. At 28 days after SCI, chronic thermal hyperalgesia was decreased significantly (by 31 to 39%; $p < 0.05$) in the pioglitazone group compared with the vehicle group. At 6 h after SCI, the pioglitazone group showed significantly less induction of inflammatory genes [interleukin (IL)-6 by 83%, IL-1 beta by 87%, monocyte chemoattractant protein-1 by 75%, intracellular adhesion molecule-1 by 84%, and early growth response-1 by 67%] compared with the vehicle group ($p < 0.05$ in all cases). Pioglitazone also significantly enhanced the post-SCI induction of neuroprotective ***heat*** ***shock*** ***proteins*** and antioxidant enzymes. Pretreatment with a PPAR gamma antagonist, 2-chloro-5-nitro-N- phenyl-benzamide (GW9662), prevented the neuroprotection induced by pioglitazone.
TI Thiazolidinedione class of peroxisome proliferator-activated receptor gamma agonists prevents neuronal damage, motor dysfunction, myelin loss, neuropathic ***pain*** , and inflammation after spinal cord injury in adult rats.
AB. . . compared with the vehicle group ($p < 0.05$ in all cases). Pioglitazone also significantly enhanced the post-SCI induction of neuroprotective ***heat*** ***shock*** ***proteins*** and antioxidant enzymes. Pretreatment with a PPAR gamma antagonist, 2-chloro-5-nitro-N- phenyl-benzamide (GW9662), prevented the neuroprotection induced by pioglitazone.
IT . . .
IT Parts, Structures, & Systems of Organisms

neuron: nervous system; spinal cord: nervous system; myelin: nervous system

IT Diseases
neuropathic ***pain*** : nervous system disease, drug therapy
Pain (MeSH)

IT Diseases
spinal cord injury: nervous system disease, injury, drug therapy
Spinal Cord Injuries (MeSH)

IT Diseases
motor dysfunction: nervous. . .

L6 ANSWER 4 OF 41 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2006:457337 BIOSIS <<LOGINID::20080330>>
DN PREV200600449882
TI Postulated vasoactive neuropeptide autoimmunity in fatigue-related conditions: A brief review and hypothesis.
AU Staines, Donald R. [Reprint Author]
CS Gold Coast Publ Hlth Unit, 10-12 Young St, Southport, Qld 4215, Australia
don_staines@health.qld.gov.au
SO Clinical & Developmental Immunology, (MAR 2006) Vol. 13, No. 1, pp. 25-39. ISSN: 1740-2522. E-ISSN: 1740-2530.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 13 Sep 2006
Last Updated on STN: 13 Sep 2006
AB Disorders such as chronic fatigue syndrome (CFS) and gulf war syndrome (GWS) are characterised by prolonged fatigue and a range of debilitating symptoms of ***pain*** , intellectual and emotional impairment, chemical sensitivities and immunological dysfunction. Sudden infant death syndrome (SIDS) surprisingly may have certain features in common with these conditions. Post-infection sequelae may be possible contributing factors although ongoing infection is unproven. Immunological aberration may prove to be associated with certain vasoactive neuropeptides (VN) in the context of molecular mimicry, inappropriate immunological memory and autoimmunity.Adenylate cyclase-activating VNs including pituitary adenylate cyclase-activating polypeptide (PACAP), vasoactive intestinal peptide (VIP) and calcitonin gene-related peptide (CGRP) act as hormones, neurotransmitters, neuroregulators, immune modulators and neurotrophic substances. They and their receptors are potentially immunogenic. VNs are widely distributed in the body particularly in the central and peripheral nervous systems and have been identified in the gut, adrenal gland, blood cells, reproductive system, lung, heart and other tissues. They have a vital role in maintaining cardio-respiratory function, thermoregulation, memory, concentration and executive functions such as emotional responses including social cues and appropriate behaviour. They are co-transmitters for a number of neurotransmitters including acetylcholine and gaseous transmitters, are potent immune regulators with primarily anti-inflammatory activity, and have a significant role in protection of the nervous system against toxic assault as well as being important in the maintenance of homeostasis.This paper describes a biologically plausible mechanism for the development of certain fatigue-related syndromes based on loss of immunological tolerance to these VNs or their receptors following infection, other events or de novo resulting in significant pathophysiology possibly mediated via CpG fragments and heat ***shock*** (stress) proteins. These conditions extend the public health context of autoimmunity and VN dysregulation and

have implications for military medicine where radiological, biological and chemical agents may have a role in pathogenesis. Possible treatment and prevention options are considered.

AB. . . fatigue syndrome (CFS) and gulf war syndrome (GWS) are characterised by prolonged fatigue and a range of debilitating symptoms of ***pain*** , intellectual and emotional impairment, chemical sensitivities and immunological dysfunction. Sudden infant death syndrome (SIDS) surprisingly may have certain features in. . . their receptors following infection, other events or de novo resulting in significant pathophysiology possibly mediated via CpG fragments and heat ***shock*** (stress) proteins. These conditions extend the public health context of autoimmunity and VN dysregulation and have implications for military medicine. . .

IT . . .
calcitonin gene-related peptide [CGRP]; adenylate cyclase [EC 4.6.1.1]; CpG; vasoactive intestinal peptide [VIP]; pituitary adenylate cyclase-activating polypeptide [PACAP]; vasoactive neuropeptide; ***heat*** ***shock*** ***proteins*** [stress proteins]

L6 ANSWER 5 OF 41 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2005:441087 BIOSIS <<LOGINID::20080330>>
DN PREV200510221627
TI Increased titres of anti-human ***heat*** ***shock*** ***protein*** 60 predict an adverse one year prognosis in patients with acute cardiac chest ***pain*** .
AU Birnie, D. H. [Reprint Author]; Vickers, L. E.; Hillis, W. S.; Norrie, J.; Cobbe, S. M.
CS Ottawa Heart Inst, Room H145,40 Ruskin Rd, Ottawa, ON K1Y 4W7, Canada
dbirnie@ottawaheart.ca
SO Heart (London), (SEP 2005) Vol. 91, No. 9, pp. 1148-1153.
ISSN: 1355-6037.
DT Article
LA English
ED Entered STN: 26 Oct 2005
Last Updated on STN: 26 Oct 2005
AB Objective: To assess whether antibodies to human ***heat*** ***shock*** ***protein*** 60 (anti-huhsp60) or to mycobacterial ***heat*** ***shock*** ***protein*** 65 (anti-mhsp65) predict an
adverse one year prognosis in patients admitted with acute cardiac chest ***pain*** .Design: Prospective observational study.Setting: Teaching hospital.Patients: 588 consecutive emergency admissions of patients with acute chest ***pain*** of suspected cardiac origin.Main outcome measures: Anti-huhsp60 and anti-mhsp65 titres were assayed on samples drawn on the morning after admission. The end points after discharge were coronary heart disease death, non-fatal myocardial infarction, coronary artery bypass grafting, percutaneous transluminal coronary angioplasty, angiogram, or readmission with further cardiac ischaemic chest ***pain*** .Results: During follow up after discharge (mean of 304 days, range 1-788 days), 277 patients had at least one of the study outcomes. Patients with increased titres of anti-huhsp60 had an adverse prognosis (hazard ratio 1.56 (95% confidence interval 1.09 to 2.23) comparing highest versus lowest quartiles, p = 0.015). Anti-mhsp65 titres were not predictive.Conclusions: Patients admitted with acute cardiac chest ***pain*** and increased titres of anti-huhsp60 had an adverse one year prognosis.
TI Increased titres of anti-human ***heat*** ***shock***

protein 60 predict an adverse one year prognosis in patients with acute cardiac chest ***pain*** .

AB Objective: To assess whether antibodies to human ***heat*** ***shock*** ***protein*** 60 (anti-huhsp60) or to mycobacterial ***heat*** ***shock*** ***protein*** 65 (anti-mhsp65) predict an adverse one year prognosis in patients admitted with acute cardiac chest ***pain*** .Design: Prospective observational study.Setting: Teaching hospital.Patients: 588 consecutive emergency admissions of patients with acute chest ***pain*** of suspected cardiac origin.Main outcome measures: Anti-huhsp60 and anti-mhsp65 titres were assayed on samples drawn on the morning after admission.. . . death, non-fatal myocardial infarction, coronary artery bypass grafting, percutaneous transluminal coronary angioplasty, angiogram, or readmission with further cardiac ischaemic chest ***pain*** .Results: During follow up after discharge (mean of 304 days, range 1-788 days), 277 patients had at least one of the. . . 2.23) comparing highest versus lowest quartiles, p = 0.015). Anti-mhsp65 titres were not predictive.Conclusions: Patients admitted with acute cardiac chest ***pain*** and increased titres of anti-huhsp60 had an adverse one year prognosis.

IT . . .
Disease (MeSH)

IT Diseases
myocardial infarction: heart disease, vascular disease, diagnosis, surgery, symptom
Myocardial Infarction (MeSH)

IT Chemicals & Biochemicals
antibodies; ***heat*** ***shock*** ***protein*** 65 [hsp65]; ***heat*** ***shock*** ***protein*** 60 [hsp60]

L6 ANSWER 6 OF 41 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AN 2005:348648 BIOSIS <<LOGINID::20080330>>

DN PREV200510142428

TI Comparative proteomic analysis of the rat spinal cord in inflammatory and neuropathic ***pain*** models.

AU Kunz, Susanne; Tegeder, Irmgard; Coste, Ovidiu; Marian, Claudiu; Pfenninger, Anja; Corvey, Carsten; Karas, Michael; Geisslinger, Gerd; Niederberger, Ellen [Reprint Author]

CS Univ Frankfurt Klinikum, Pharmazentrum Frankfurt, ZAFES, Theodor Stern Kai 7, D-60590 Frankfurt, Germany
e.niederberger@em.uni-frankfurt.de

SO Neuroscience Letters, (JUN 24 2005) Vol. 381, No. 3, pp. 289-293.
CODEN: NELED5. ISSN: 0304-3940.

DT Article

LA English

ED Entered STN: 8 Sep 2005
Last Updated on STN: 8 Sep 2005

AB Pathological ***pain*** associated either with peripheral tissue damage and inflammation (inflammatory ***pain***) or peripheral nerve injury (neuropathic ***pain***) is characterized by persistent ***pain*** hypersensitivity. This hypersensitivity is believed to be mediated by sensitization of nociceptors and spinal dorsal horn neurons leading to hyperalgesia and allodynia. Changes of protein expression and/or phosphorylation are known to contribute to the development of this hyperexcitability of the nociceptive system. In the present study we analyzed protein patterns in the spinal cord following paw inflammation or sciatic nerve injury using two-dimensional (2D) gel electrophoresis

combined with MALDI-TOF mass spectrometry. 2D-PAGE revealed nine and five regulated proteins following paw inflammation and sciatic nerve damage, respectively. These regulated proteins had not been identified previously with other methods. There was no overlap of regulated proteins between models except for the small ***heat*** ***shock*** ***protein*** alpha-crystallin, which was decreased in both models. In conclusion, this study illustrates that employment of the proteornic 2D-PAGE approach allows for identification of novel regulated proteins that may be involved in the central sensitization and possibly manifestation of chronic ***pain*** . (c) 2005 Elsevier Ireland Ltd. All rights reserved.

TI Comparative proteomic analysis of the rat spinal cord in inflammatory and neuropathic ***pain*** models.

AB Pathological ***pain*** associated either with peripheral tissue damage and inflammation (inflammatory ***pain***) or peripheral nerve injury (neuropathic ***pain***) is characterized by persistent ***pain*** hypersensitivity. This hypersensitivity is believed to be mediated by sensitization of nociceptors and spinal dorsal horn neurons leading to hyperalgesia. . . not been identified previously with other methods. There was no overlap of regulated proteins between models except for the small ***heat*** ***shock*** ***protein*** alpha-crystallin, which was decreased in both models. In conclusion, this study illustrates that employment of the proteornic 2D-PAGE approach allows for identification of novel regulated proteins that may be involved in the central sensitization and possibly manifestation of chronic ***pain*** . (c) 2005 Elsevier Ireland Ltd. All rights reserved.

IT . . .
spinal cord: nervous system; spinal dorsal horn neuron: nervous system

IT Diseases
hyperalgesia: nervous system disease
Hyperalgesia (MeSH)

IT Diseases
chronic ***pain*** : nervous system disease
Pain (MeSH)

IT Diseases
sciatic nerve injury: nervous system disease, injury

IT Diseases
neuropathic ***pain*** : nervous system disease, pathology
Pain (MeSH)

IT Chemicals & Biochemicals
alpha-crystallin: small ***heat*** ***shock*** ***protein***

L6 ANSWER 7 OF 41 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AN 2005:335072 BIOSIS <<LOGINID::20080330>>

DN PREV200510121295

TI The ***heat*** ***shock*** ***protein*** 90 inhibitor, 17-allylamino-17-demethoxygeldanamycin, enhances osteoclast formation and potentiates bone metastasis of a human breast cancer cell line.

AU Price, John T. [Reprint Author]; Quinn, Julian M. W.; Sims, Natalie A.; Vieusseux, Jessica; Waldeck, Kelly; Docherty, Susan E.; Myers, Damian; Nakamura, Akira; Waltham, Mark C.; Gillespie, Matthew T.; Thompson, Erik W.

CS St Vincents Inst Med Res, Tumour Cell Migrat and Metastasis Lab, 41 Victoria Parade, Melbourne, Vic 3065, Australia
jprice@svi.edu.au

SO Cancer Research, (JUN 1 2005) Vol. 65, No. 11, pp. 4929-4938.
CODEN: CNREA8. ISSN: 0008-5472.

DT Article

LA English
ED Entered STN: 31 Aug 2005
Last Updated on STN: 31 Aug 2005
AB Breast cancer metastasis to the bone occurs frequently, causing numerous complications including severe ***pain*** , fracture, hypercalcemia, and paralysis. Despite its prevalence and severity, few effective therapies exist. To address this, we examined whether the ***heat*** ***shock*** ***protein*** 90 (Hsp90) inhibitor, 17-allylamino-17-demethoxygeldanamycin (17-AAG), would be efficacious in inhibiting breast cancer metastasis to bone. Utilizing the human breast cancer subline, MDA-MB-231SA, previously in vivo selected for its enhanced ability to generate osteolytic bone lesions, we determined that 17-AAG potently inhibited its in vitro proliferation and migration. Moreover, 17-AAG significantly reduced MDA-MB-231SA tumor growth in the mammary-fat pad of nude mice. Despite these findings, 17-AAG enhanced the incidence of bone metastasis and osteolytic lesions following intracardiac inoculation in the nude mouse. Consistent with these findings, 17-AAG enhanced osteoclast formation 2- to 4-fold in mouse bone marrow/osteoblast cocultures, receptor activator of nuclear factor kappa B ligand (RANKL)-stimulated bone marrow, and RAW264.7 cell models of in vitro osteoclastogenesis. Moreover, the drug enhanced osteoclastogenesis in human cord blood progenitor cells, demonstrating that its effects were not limited to mouse models. In addition to 17-AAG, other Hsp90 inhibitors, such as radicicol and herbimycin A, also enhanced osteoclastogenesis. A pro-osteolytic action of 17-AAG independent of tumor presence was also determined in vivo, in which 17-AAG-treated tumor-naive mice had reduced trabecular bone volume with an associated increase in osteoclast number. Thus, HSP90 inhibitors can stimulate osteoclast formation, which may underlie the increased incidence of osteolysis and skeletal tumor incidence causedby 17-AAG in vivo. These data suggest an important contraindication to the Hsp90 targeted cancer therapy currently undergoing clinical trial.
TI The ***heat*** ***shock*** ***protein*** 90 inhibitor, 17-allylamino-17-demethoxygeldanamycin, enhances osteoclast formation and potentiates bone metastasis of a human breast cancer cell line.
AB Breast cancer metastasis to the bone occurs frequently, causing numerous complications including severe ***pain*** , fracture, hypercalcemia, and paralysis. Despite its prevalence and severity, few effective therapies exist. To address this, we examined whether the ***heat*** ***shock*** ***protein*** 90 (Hsp90) inhibitor, 17-allylamino-17-demethoxygeldanamycin (17-AAG), would be efficacious in inhibiting breast cancer metastasis to bone. Utilizing the human breast cancer. . .
IT . . .
disease, drug therapy
Bone Neoplasms (MeSH); Neoplasm Metastasis (MeSH)
IT Diseases
hypercalcemia: metabolic disease, drug therapy
Hypercalcemia (MeSH)
IT Chemicals & Biochemicals
heat ***shock*** ***protein*** 90 [Hsp90]; receptor activator of nuclear factor kappa B ligand [RANKL]; radicicol: antineoplastic-drug; 17-allylamino-17-demethoxygeldanamycin [17-AAG]: antineoplastic-drug; herbimycin A: antineoplastic-drug

L6 ANSWER 8 OF 41 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2005:99877 BIOSIS <<LOGINID::20080330>>
DN PREV200500097576

TI Exacerbation of rheumatoid arthritis following Helicobacter pylori eradication: disruption of established oral tolerance against ***heat*** ***shock*** ***protein*** ?.
AU Matsukawa, Yoshihiro [Reprint Author]; Asai, Yasukiyo; Kitamura, Noboru; Sawada, Shigemasa; Kurosaka, Hanzo
CS Sch MedDiv Hematol and RheumatolDept Med, Nihon Univ, Tokyo, 1738610, Japan
m-2000@mbk.ocn.ne.jp
SO Medical Hypotheses, (2005) Vol. 64, No. 1, pp. 41-43. print.
ISSN: 0306-9877 (ISSN print).
DT Article
LA English
ED Entered STN: 9 Mar 2005
Last Updated on STN: 9 Mar 2005
AB A 62-year-old Japanese woman with RA received an eradication therapy against Helicobacter pylori in November 1999. Eight weeks Later, successful eradication was confirmed by negative results for rapid urease test, pathologic findings, and a fall in anti-H. pylori IgG antibody titer. During the course, parameters for RA activity were exacerbated: C-reactive protein 1.1-4.2 mg/dL, rheumatoid arthritis precipitation antigen 25605120 dils., erythrocyte sedimentation rate 52-123 mm/h, and complements CH50 50 to over 60 U/mL. Lansbury index increased from 70% to 105%. Two more weeks later, the patient noticed right shoulder ***pain*** . She also complained of bilateral gonalgia two months later, and physical examination revealed increased fluid in the knee joints. Prednisolone was required to control the disease activity. The results of this case suggested that RA patients might experience a deleterious effect on the disease activity following H. pylori eradication possibly through disruption of the established oral tolerance against stress protein such as mycobacterial ***heat*** ***shock*** ***protein*** 65.

TI Exacerbation of rheumatoid arthritis following Helicobacter pylori eradication: disruption of established oral tolerance against ***heat*** ***shock*** ***protein*** ?.
AB. . . to over 60 U/mL. Lansbury index increased from 70% to 105%. Two more weeks later, the patient noticed right shoulder ***pain*** . She also complained of bilateral gonalgia two months later, and physical examination revealed increased fluid in the knee joints. Prednisolone. . . disease activity following H. pylori eradication possibly through disruption of the established oral tolerance against stress protein such as mycobacterial ***heat*** ***shock*** ***protein*** 65.

IT . . .
rheumatoid arthritis: connective tissue disease, immune system disease, joint disease, therapy
Arthritis, Rheumatoid (MeSH)
IT Chemicals & Biochemicals
c-reactive protein; mycobacterial ***heat*** ***shock*** ***protein*** 65; prednisolone: antiarthritic-drug, antiinflammatory-drug, immunologic-drug, immunosuppressant-drug

L6 ANSWER 9 OF 41 BIOSIS
AN 2005:19890 BIOSIS <<LOGINID::20080330>>
DN PREV200500017849
TI Doxycycline versus doxycycline and rifampin in undifferentiated spondyloarthropathy, with special reference to Chlamydia-Induced

arthritis. a prospective, randomized 9-month comparison.
AU Carter, John D. [Reprint Author]; Valeriano, Joanne; Vasey, Frank B.
CS Div Rheumatol, Univ S Florida, 12901 Bruce B Downs Blvd,MDC 81, Tampa, FL, 33612, USA
jocarter@hsc.us.fedu
SO Journal of Rheumatology, (October 2004) Vol. 31, No. 10, pp. 1973-1980. print.
ISSN: 0315-162X (ISSN print).
DT Article
LA English
ED Entered STN: 22 Dec 2004
Last Updated on STN: 22 Dec 2004
AB Objective. Chlamydia is a known trigger of reactive arthritis (ReA). It may also be common cause of undifferentiated spondyloarthropathy (uSpA). Persistent, metabolically active, Chlamydiae have been observed in the synovial tissue of these patients years after their initial exposure. Trials with lymecycline and rifampin have shown benefit in early/acute Chlamydia-induced arthritis. In vitro data suggest that persistent Chlamydia become resistant to chronic monotherapy of tetracyclines or rifampin, whereas no such resistance is noted when rifampin is added to antimicrobials that are active against Chlamydia. Rifampin and doxycycline also show synergistic effect against Chlamydia. In addition, rifampin inhibits chlamydial production of ***heat*** ***shock*** ***proteins*** (HSP). HSP60 plays a key role in the chronic persistent state of Chlamydia. We conducted a prospective, randomized 9-month trial to evaluate the efficacy of doxycycline versus a combination of doxycycline and rifampin in the treatment of uSpA. Methods. The study enrolled 30 patients with chronic inflammatory arthritis (average disease duration 10 yrs) who fulfilled the European Spondylarthropathy Study Group criteria, with no evidence of inflammatory bowel disease, psoriasis, ankylosing spondylitis, or preceding dysentery. Patients received doxycycline 100 mg po twice daily or a combination of doxycycline 100 rug po twice daily and rifampin 600 mg po daily. They received a 4-question self-questionnaire and a blinded joint examination at each visit. The questions include a visual analog scale (VAS) for their current amount of back ***pain*** , duration of morning stiffness, back ***pain*** at night, and peripheral joint ***pain*** . The blinded joint examination consisted of a swollen joint count (SJC) and a tender joint count (TIC). These 6 variables were assessed at baseline and at 1, 3, 6, and 9 months. Responders were defined as those who improved gtoreq 20% in at least 4 of the 6 variables at 9 months of therapy. Results. Comparing the doxycycline + rifampin arm (D/R) versus the doxycycline arm (D) at 9 months of therapy, all 6 variables improved more in D/R versus D, 4 of which were statistically significant. The mean VAS (scale of 100) decreased 24.4 points in D/R in contrast to 3 points in D ($p < 0.03$). Duration of morning stiffness decreased by 1.2 h in D/R, with a slight increase of 0.1 h in D ($p < 0.003$). The back ***pain*** at night and peripheral joint ***pain*** both improved in D/R group versus D (not statistically significant). Finally, the SJC and TJC also improved in D/R (-2.1 and -2.5) versus D (-0.4 and -0.6; $p = 0.02$, $p = 0.03$, respectively). Eleven of 15 patients in the D/R arm were responders, whereas only 2 of 15 D group patients were considered responders ($p < 0.003$). Conclusion. The combination of doxycycline and rifampin for 9 months seemed to be effective in treatment of chronic uSpA. This is the first study to demonstrate therapeutic benefit with antimicrobials to a chronic inflammatory arthritis possibly secondary to persistent Chlamydia.
AB. . . are active against Chlamydia. Rifampin and doxycycline also show

synergistic effect against Chlamydia. In addition, rifampin inhibits chlamydial production of ***heat*** ***shock*** ***proteins*** (HSP). HSP60 plays a key role in the chronic persistent state of Chlamydia. We conducted a prospective, randomized 9-month trial. . . blinded joint examination at each visit. The questions include a visual analog scale (VAS) for their current amount of back ***pain*** , duration of morning stiffness, back ***pain*** at night, and peripheral joint ***pain*** . The blinded joint examination consisted of a swollen joint count (SJC) and a tender joint count (TIC). These 6 variables. . . decreased by 1.2 h in D/R, with a slight increase of 0.1 h in D ($p < 0.003$). The back ***pain*** at night and peripheral joint ***pain*** both improved in D/R group versus D (not statistically significant). Finally, the SJC and TJC also improved in D/R (-2.1. . .

IT . . .
undifferentiated spondyloarthropathy: immune system disease, joint disease, drug therapy, etiology, chronic inflammatory arthritis

IT Chemicals & Biochemicals
doxycycline: antibacterial-drug, antiinfective-drug; ***heat*** ***shock*** ***protein*** ; lymecycline: antibacterial-drug, antiinfective-drug; rifampin: antibacterial-drug, antiinfective-drug, enzyme inhibitor-drug; tetracycline: antibacterial-drug, antiinfective-drug, enzyme inhibitor-drug

L6 ANSWER 10 OF 41 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AN 2004:146512 BIOSIS <<LOGINID::20080330>>

DN PREV200400146331

TI Changes in spinal cord gene expression in a mouse model of cancer ***pain*** .

AU Baddorf, M. J. [Reprint Author]; Kehl, L. J.; Lynch, J. L. [Reprint Author]; Eikmeier, L. J. [Reprint Author]; Beitz, A. J. [Reprint Author]

CS Vet. PathoBiol., Univ. of Minnesota, St. Paul, MN, USA

SO Society for Neuroscience Abstract Viewer and Itinerary Planner, (2003) Vol. 2003, pp. Abstract No. 815.11. http://sfn.scholarone.com. e-file. Meeting Info.: 33rd Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 08-12, 2003. Society of Neuroscience.

DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LA English

ED Entered STN: 17 Mar 2004
Last Updated on STN: 17 Mar 2004

AB It is well appreciated that the greatest impact on the quality of life of patients with malignant disease is cancer ***pain*** . Cancer ***pain*** , like many other forms of chronic ***pain*** , is thought to be associated with both biochemical and functional changes in the spinal cord. To study the central mechanisms underlying tumor-induced ***pain*** , we have used a mouse tumor model to investigate changes in gene expression occurring in the spinal cord at the peak of tumor-induced hyperalgesia. This model involves implantation of fibrosarcoma cells into the mouse calcaneous bone with subsequent mechanical hyperalgesia appearing at day 3 and peaking at days 10-13 post-implantation. To characterize the changes in spinal cord gene expression associated with the development of cancer ***pain*** , Affymetrix Murine U74Av2 microarrays were used in triplicate to analyze spinal cord mRNA from tumor-bearing and naive male mice. Spinal cord samples from tumor-bearing mice were collected at day 12 post-implantation, and only mice showing significant primary hyperalgesia were included in the analysis. GeneData

Expressionist was used to perform replicate analysis, and the data was statistically analyzed using a t-test with a Welch correction to identify genes that were differentially expressed between the two groups. Transcriptional changes in the spinal cords of fibrosarcoma-implanted mice included a significant upregulation of a number of genes including thyrotropin releasing hormone receptor, the activin beta-A subunit, and vinculin, as well as a significant downregulation of a number of genes including several ***heat*** ***shock*** ***proteins*** . The results indicate a number of spinal cord genes are up-or down-regulated in tumor-bearing mice compared to controls and it is feasible that many of these genes play a role in the development of the unique ***pain*** state associated with malignant disease.

TI Changes in spinal cord gene expression in a mouse model of cancer ***pain*** .

AB. . . It is well appreciated that the greatest impact on the quality of life of patients with malignant disease is cancer ***pain*** . Cancer ***pain*** , like many other forms of chronic ***pain*** , is thought to be associated with both biochemical and functional changes in the spinal cord. To study the central mechanisms underlying tumor-induced ***pain*** , we have used a mouse tumor model to investigate changes in gene expression occurring in the spinal cord at the. . . peaking at days 10-13 post-implantation. To characterize the changes in spinal cord gene expression associated with the development of cancer ***pain*** , Affymetrix Murine U74Av2 microarrays were used in triplicate to analyze spinal cord mRNA from tumor-bearing and naive male mice. Spinal. . . receptor, the activin beta-A subunit, and vinculin, as well as a significant downregulation of a number of genes including several ***heat*** ***shock*** ***proteins*** . The results indicate a number of spinal cord genes are up-or down-regulated in tumor-bearing mice compared to controls and it is feasible that many of these genes play a role in the development of the unique ***pain*** state associated with malignant disease.

IT . . .
Medicine, Medical Sciences)

IT Parts, Structures, & Systems of Organisms
bone: skeletal system; spinal cord: nervous system

IT Diseases
cancer ***pain*** : neoplastic disease, nervous system disease

IT Diseases
chronic ***pain*** : nervous system disease
Pain (MeSH)

IT Diseases
fibrosarcoma: neoplastic disease
Fibrosarcoma (MeSH)

IT Diseases
hyperalgesia: nervous system disease
Hyperalgesia (MeSH)

IT Diseases
mechanical hyperalgesia: nervous system disease
Hyperalgesia (MeSH)

IT Diseases
pain : nervous system disease
Pain (MeSH)

IT Diseases
pain state: nervous system disease

IT Diseases
primary hyperalgesia: nervous system disease

IT Chemicals & Biochemicals
activin; genes; ***heat*** ***shock*** ***protein*** ; mRNA; thyrotropin-releasing hormone receptor; vinculin

L6 ANSWER 11 OF 41 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2004:125526 BIOSIS <<LOGINID::20080330>>
DN PREV200400125165
TI Review of NMDA antagonist-induced neurotoxicity and implications for clinical development.
AU Low, S. J. [Reprint Author]; Roland, C. L.
CS Cognetix, Inc., 421 Wakara Way, Suite 201, Salt Lake City, UT, 84108, USA
SO International Journal of Clinical Pharmacology and Therapeutics, (January 2004) Vol. 42, No. 1, pp. 1-14. print.
ISSN: 0946-1965.
DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 3 Mar 2004
Last Updated on STN: 3 Mar 2004
AB NMDA receptor antagonists have been investigated for many years as therapeutic agents for the treatment of neurological disorders such as stroke, epilepsy, ***pain*** and Parkinson's disease. It has been discovered, however, that many of these compounds cause adverse behavioral (psychotomimetic) effects and can produce neurotoxicity characterized by neuronal vacuolization, induction of ***heat*** - ***shock*** ***protein*** , neuronal/axonal degeneration and regional brain cell death in several animal species. It is unknown whether NMDA antagonists induce neurotoxicity in humans. The mechanism of NMDA antagonist-induced neurotoxicity is not completely known, but some evidence suggests disinhibition of GABAergic inputs to the affected neurons. Several classes of compounds have been shown to prevent NMDA antagonist-induced neurotoxicity. The extent of neurotoxicity produced by NMDA antagonists is affected by many factors, including type of antagonist, dose, length of exposure, age, sex and species. While there are no published regulatory guidelines regarding how NMDA antagonist compounds should be evaluated, sponsors and investigators of these compounds should make every effort to assess the potential for neurotoxicity. NMDA receptor antagonists, as well as other CNS-active compounds need to be analyzed for neurotoxicity through careful experimental design, adequate tissue sampling and through the use of a sensitive method of detection.
AB. . . antagonists have been investigated for many years as therapeutic agents for the treatment of neurological disorders such as stroke, epilepsy, ***pain*** and Parkinson's disease. It has been discovered, however, that many of these compounds cause adverse behavioral (psychotomimetic) effects and can produce neurotoxicity characterized by neuronal vacuolization, induction of ***heat*** - ***shock*** ***protein*** , neuronal/axonal degeneration and regional brain cell death in several animal species. It is unknown whether NMDA antagonists induce neurotoxicity in. . .
IT . . .
Disorders (MeSH)
IT Chemicals & Biochemicals
NMDA receptor antagonist: NMDA receptor antagonist-drug, clinical development, neurotoxin; dextromethorphan: NMDA receptor antagonist-drug; glutamate; ***heat*** ***shock*** ***protein*** ; ketamine: NMDA receptor antagonist-drug

L6 ANSWER 12 OF 41 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2004:112150 BIOSIS <<LOGINID::20080330>>
DN PREV200400113030
TI The effect of treatment with BRX-220, a co-inducer of ***heat*** ***shock*** ***proteins*** , on sensory fibers of the rat following peripheral nerve injury.
AU Kalmar, B. [Reprint Author]; Greensmith, L.; Malcangio, M.; McMahon, S. B.; Csermely, P.; Burnstock, G.
CS Sobell Department of Motor Neuroscience and Movement Disorders, Institute of Neurology, Queen Square, London, WC1N 3BG, UK
b.kalmar@ucl.ac.uk
SO Experimental Neurology, (December 2003) Vol. 184, No. 2, pp. 636-647. print.
CODEN: EXNEAC. ISSN: 0014-4886.
DT Article
LA English
ED Entered STN: 25 Feb 2004
Last Updated on STN: 25 Feb 2004
AB In this study, we examined the effect BRX-220, a co-inducer of ***heat*** ***shock*** ***proteins*** , in injury-induced peripheral neuropathy. Following sciatic nerve injury in adult rats and treatment with BRX-220, the following features of the sensory system were studied: (a) expression of calcitonin gene-related peptide (CGRP); (b) binding of isolectin B4 (IB4) in dorsal root ganglia (DRG) and spinal cord; (c) stimulation-evoked release of substance P (SP) in an in vitro spinal cord preparation and (d) nociceptive responses of partially denervated rats. BRX-220 partially reverses axotomy-induced changes in the sensory system. In vehicle-treated rats there is a decrease in IB4 binding and CGRP expression in injured neurones, while in BRX-220-treated rats these markers were better preserved. Thus, 7.0+-0.6% of injured DRG neurones bound IB4 in vehicle-treated rats compared to 14.4+-0.9% in BRX-220-treated animals. Similarly, 4.5+-0.5% of DRG neurones expressed CGRP in the vehicle-treated group, whereas 9.0+-0.3% were positive in the BRX-220-treated group. BRX-220 also partially restored SP release from spinal cord sections to electrical stimulation of primary sensory neurones. Behavioural tests carried out on partially denervated animals showed that BRX-220 treatment did not prevent the emergence of mechanical or thermal hyperalgesia. However, oral treatment for 4 weeks lead to reduced ***pain*** -related behaviour suggesting either slowly developing analgesic actions or enhancement of recovery processes. Thus, the morphological improvement seen in sensory neurone markers was accompanied by restored functional activity. Therefore, treatment with BRX-220 promotes restoration of morphological and functional properties in the sensory system following peripheral nerve injury.
TI The effect of treatment with BRX-220, a co-inducer of ***heat*** ***shock*** ***proteins*** , on sensory fibers of the rat following peripheral nerve injury.
AB In this study, we examined the effect BRX-220, a co-inducer of ***heat*** ***shock*** ***proteins*** , in injury-induced peripheral neuropathy. Following sciatic nerve injury in adult rats and treatment with BRX-220, the following features of the. . . treatment did not prevent the emergence of mechanical or thermal hyperalgesia. However, oral treatment for 4 weeks lead to reduced ***pain*** -related behaviour suggesting either slowly developing analgesic actions or enhancement of recovery processes. Thus, the morphological improvement

seen in sensory neurone. . .

L6 ANSWER 13 OF 41 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2004:14294 BIOSIS <<LOGINID::20080330>>
DN PREV200400015911
TI The toxicogenomics of abacavir hypersensitivity.
AU Mallal, Simon [Reprint Author]
CS Ctr. for Clin. Immunology and Biomedical Statistics, Perth, WA, Australia
SO Abstracts of the Interscience Conference on Antimicrobial Agents and Chemotherapy, (2003) Vol. 43, pp. 520. print.
Meeting Info.: 43rd Annual Interscience Conference on Antimicrobial Agents and Chemotherapy. Chicago, IL, USA. September 14-17, 2003. American Society for Microbiology.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 24 Dec 2003
Last Updated on STN: 24 Dec 2003
AB Abacavir hypersensitivity is a potentially life-threatening syndrome affecting 1-14% of abacavir-exposed individuals (average apprx4%), manifesting as fever, rash, gastrointestinal symptoms (nausea, vomiting, diarrhoea or abdominal ***pain***), with less common respiratory, renal, hepatic or musculoskeletal involvement. Symptoms usually appear within the first six weeks of treatment, worsen with continued therapy and usually improve within 24 hours of abacavir discontinuation. Rechallenging with abacavir following a hypersensitivity reaction typically results in recurrence of symptoms within hours, with the potential to induce a more severe clinical syndrome with increased risk of life-threatening hypotension and death. Genetically determined susceptibility to this syndrome was initially suggested by clinical observations that only a subset of individuals exposed to abacavir develop hypersensitivity whilst those who are initially tolerant remain at low risk despite ongoing therapy; and that black racial origin was associated with significantly reduced risk in a meta-analysis of 25 clinical studies involving 5248 subjects. Subsequently, two independent studies have demonstrated that specific MHC alleles are strongly associated with risk of abacavir hypersensitivity, with an independent positive predictive value of greater than 70% (assuming equivalent prevalence of the hypersensitivity syndrome in the study populations) associated with the presence of the HLA-B*5701 allele. Racial differences in the prevalence of abacavir hypersensitivity also mirror the known HLA-B*5701 phenotype frequency, although transracial studies confirming this association are still required. Prospective experience with HLA typing in our cohort (n=48) has confirmed the utility of genetic testing, with a combined analysis of abacavir-exposed individuals (n=248) revealing 18 cases of abacavir hypersensitivity (confirmed with epicutaneous patch testing and/or ex vivo immunological studies), and 230 abacavir tolerant individuals. Of these, HLA-B*5701 was present in 94% of cases and 1.7% of controls (OR 587, PV+ 81%, PV- 99.6%). Based on these data, prospective testing for HLA-B*5701 carriage would be predicted to reduce abacavir HSR incidence from 7.3% to 0.4%, while inappropriately denying 1.6% of the population access to abacavir. In terms of cost-effectiveness, it would be predicted that apprx14 individuals would need to be tested to prevent 1 case of abacavir hypersensitivity, at a cost of apprxdollar sign280 (given that molecular methods for specific HLA-B*5701 typing cost apprxdollar sign20 per test). Presence of HLA-B*5701 and a central MHC polymorphism

was found in 94% of cases and 0.43% of controls (OR 3910, PV+ 94%, PV- 99.5%), with recombinant genetic mapping suggesting a susceptibility locus within the highly conserved ***heat*** ***shock*** ***protein*** 70 (hsp70) gene cluster. However, these data suggest that the HLA-B*5701 provides specificity to the abacavir-induced immune response, in that the HLA-B*5701 allele is necessary but not sufficient in isolation, for genetic susceptibility. Consistent with this possibility, depletion of CD8+ cells resulted in marked attenuation of abacavir-stimulated TNF-alpha expression ex vivo, suggesting a Class I-restricted immune response to abacavir. Whilst these data suggest that typing for selected HLA alleles provides a highly predictive and cost-effective test for abacavir hypersensitivity (at least among Caucasian populations) there are a number of impediments to widespread uptake of genetic testing, which will be discussed.

AB. . . life-threatening syndrome affecting 1-14% of abacavir-exposed individuals (average apprx4%), manifesting as fever, rash, gastrointestinal symptoms (nausea, vomiting, diarrhoea or abdominal ***pain***), with less common respiratory, renal, hepatic or musculoskeletal involvement. Symptoms usually appear within the first six weeks of treatment, worsen. . . of controls (OR 3910, PV+ 94%, PV- 99.5%), with recombinant genetic mapping suggesting a susceptibility locus within the highly conserved ***heat*** ***shock*** ***protein*** 70 (hsp70) gene cluster. However, these data suggest that the HLA-B*5701 provides specificity to the abacavir-induced immune response, in that. . .

IT . . .
drug hypersensitivity: immune system disease
Drug Hypersensitivity (MeSH)

IT Chemicals & Biochemicals
HLA; TNF-alpha [tumor necrosis factor-alpha]: expression; abacavir: antiinfective-drug, antiviral-drug; ***heat*** ***shock*** ***protein*** 70

GEN human HLA gene (Hominidae): B allele; human hsp70 gene [human ***heat*** ***shock*** ***protein*** 70 gene] (Hominidae)

L6 ANSWER 14 OF 41 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AN 2003:571255 BIOSIS <<LOGINID::20080330>>

DN PREV200300575078

TI Prevalence of intimal pathogen burden in acute coronary syndromes. Original Title: Praevalenz von intimalem Pathogen burden bei akutem Koronarsyndrom..

AU Andrie, R. [Reprint Author]; Braun, P.; Heinrich, K.-W.; Luederitz, B.; Bauriedel, G.

CS Medizinische Klinik und Poliklinik II, Universitaetsklinikum Bonn, Sigmund-Freud-Str. 25, 53105, Bonn, Germany
R.Andrei@gmx.de

SO Zeitschrift fuer Kardiologie, (August 2003) Vol. 92, No. 8, pp. 641-649. print.
CODEN: ZKRDAX. ISSN: 0300-5860.

DT Article

LA German

ED Entered STN: 10 Dec 2003
Last Updated on STN: 10 Dec 2003

AB Increasing evidence supports a link between serological evidence of prior exposure to infectious pathogens, pathogen burden, and the risk for future myocardial infarction and death in patients with coronary artery disease.

Based on this concept, we evaluated the intimal presence of four pathogens in human coronary atheroma, clinically associated with acute coronary syndromes (ACS) and stable angina (SA), and the effect of pathogen burden on the expression of human ***heat*** - ***shock*** ***protein*** 60 (hHSP60), a key protein in (auto-)immune pathogenesis of atherosclerosis. Coronary atherectomy specimens retrieved from 53 primary target lesions of patients with ACS (n=33) or SA (n=20) were assessed immunohistochemically for the presence of Chlamydia pneumoniae (C.pn.), Helicobacter pylori (H.p.), Cytomegalovirus (CMV) and Epstein-Barr Virus (EBV), and for the expression of hHSP60. Chlamydia pneumoniae was present in 74%, Helicobacter pylori in 32%, CMV in 13% and EBV in 42%. Exclusively C.pn. revealed a prevalence in ACS (91%) vs SA (45%; $p < 0.001$). Immunohistochemical analysis revealed 6 lesions without, 21 lesions with 1, 17 lesions with 2, 6 lesions with 3 and 3 lesions with 4 infectious agents. As an important finding, the mean value in ACS lesions was significantly increased compared to those in SA (1.9 vs 1.1; $p < 0.01$). ACS-subgroup analysis revealed the highest mean value in patients with ***pain*** at rest within the last two days (Braunwald class III). In addition, expression of hHSP60 was significantly higher in ACS (8.7%) compared to SA (1.3%; $p < 0.001$). Pathogen burden correlated highly significant ($p < 0.01$) with the expression of hHSP60 ($r = 0.44$). Our data demonstrate the impact of intimal pathogen burden in plaque instability, and suggest the presence of (auto-)immunoreactions against upregulated hHSP60 as an important pathomechanism that may contribute to acute coronary syndromes.

AB. . . with acute coronary syndromes (ACS) and stable angina (SA), and the effect of pathogen burden on the expression of human ***heat*** - ***shock*** ***protein*** 60 (hHSP60), a key protein in (auto-)immune pathogenesis of atherosclerosis. Coronary atherectomy specimens retrieved from 53 primary target lesions of. . . to those in SA (1.9 vs 1.1; $p < 0.01$). ACS-subgroup analysis revealed the highest mean value in patients with ***pain*** at rest within the last two days (Braunwald class III). In addition, expression of hHSP60 was significantly higher in ACS. . .

IT . . .
cytomegalovirus infection: viral disease, pathology
Cytomegalovirus Infections (MeSH)

IT Diseases
stable angina: heart disease, pathology
Angina Pectoris (MeSH)

IT Chemicals & Biochemicals
heat - ***shock*** ***protein*** 60

L6 ANSWER 15 OF 41 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AN 2002:526413 BIOSIS <<LOGINID::20080330>>

DN PREV200200526413

TI Molecular adaptations of neuromuscular disease-associated proteins in response to eccentric exercise in human skeletal muscle.

AU Feasson, L. [Reprint author]; Stockholm, D.; Freyssenet, D.; Richard, I.; Duguez, S.; Beckmann, J. S.; Denis, C.

CS Laboratoire de Physiologie-Groupe PPEH GIP E25, Faculte de Medecine, 15 rue Ambroise Pare, 42023, Saint-Etienne, France
Leonard.Feasson@univ-st-etienne.fr

SO Journal of Physiology (Cambridge), (15 August, 2002) Vol. 543, No. 1, pp. 297-306. print.
CODEN: JPHYA7. ISSN: 0022-3751.

DT Article
LA English
ED Entered STN: 9 Oct 2002
Last Updated on STN: 9 Oct 2002
AB The molecular events by which eccentric muscle contractions induce muscle damage and remodelling remain largely unknown. We assessed whether eccentric exercise modulates the expression of proteinases (calpains 1, 2 and 3, proteasome, cathepsin B+L), muscle structural proteins (alpha-sarcoglycan and desmin), and the expression of the ***heat*** ***shock*** ***proteins*** Hsp27 and alphaB-crystallin. Vastus lateralis muscle biopsies from twelve healthy male volunteers were obtained before, immediately after, and 1 and 14 days after a 30 min downhill treadmill running exercise. Eccentric exercise induced muscle damage as evidenced by the analysis of muscle ***pain*** and weakness, creatine kinase serum activity, myoglobinaemia and ultrastructural analysis of muscle biopsies. The calpain 3 mRNA level was decreased immediately after exercise whereas calpain 2 mRNA level was increased at day 1. Both mRNA levels returned to control values by day 14. By contrast, cathepsin B+L and proteasome enzyme activities were increased at day 14. The alpha-sarcoglycan protein level was decreased immediately after exercise and at day 1, whereas the desmin level peaked at day 14. alphaB-crystallin and Hsp27 protein levels were increased at days 1 and 14. Our results suggest that the differential expression of calpain 2 and 3 mRNA levels may be important in the process of exercise-induced muscle damage, whereas expression of alpha-sarcoglycan, desmin, alphaB-crystallin and Hsp27 may be essentially involved in the subsequent remodelling of myofibrillar structure. This remodelling response may limit the extent of muscle damage upon a subsequent mechanical stress.
AB. . . proteinases (calpains 1, 2 and 3, proteasome, cathepsin B+L), muscle structural proteins (alpha-sarcoglycan and desmin), and the expression of the ***heat*** ***shock*** ***proteins*** Hsp27 and alphaB-crystallin. Vastus lateralis muscle biopsies from twelve healthy male volunteers were obtained before, immediately after, and 1 and. . . after a 30 min downhill treadmill running exercise. Eccentric exercise induced muscle damage as evidenced by the analysis of muscle ***pain*** and weakness, creatine kinase serum activity, myoglobinaemia and ultrastructural analysis of muscle biopsies. The calpain 3 mRNA level was decreased. . .
IT . . .
system
IT Chemicals & Biochemicals
alpha-B-crystallin; alpha-sarcoglycan; calpain 1; calpain 2; calpain 3; cathepsin B; cathepsin L; creatine kinase; desmin; ***heat*** ***shock*** ***protein*** 27; mRNA [messenger RNA]; myoglobin; neuromuscular disease-associated proteins: molecular adaptations; proteasome

L6 ANSWER 16 OF 41 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2002:7123 BIOSIS <<LOGINID::20080330>>
DN PREV200200007123
TI Differentially expressed genes in rat dorsal root ganglia following peripheral nerve injury.
AU Kim, Dong-Sun; Lee, Sang-Ji; Park, So-Yun; Yoo, Hea-Jin; Kim, Shin-Hee; Kim, Kwang-Jin; Cho, Hee-Jung [Reprint author]
CS Department of Anatomy, School of Medicine, Kyungpook National University, 2-101, Dongin Dong, Taegu, 700-422, South Korea

SO Neuroreport, (29 October, 2001) Vol. 12, No. 15, pp. 3401-3405. print.
CODEN: NERPEZ. ISSN: 0959-4965.
DT Article
LA English
ED Entered STN: 28 Dec 2001
Last Updated on STN: 25 Feb 2002
AB Ordered differential display PCR was used to identify differentially expressed genes in rat dorsal root ganglia at 7 days following chronic constriction injury (CCI) of the sciatic nerve. Fourteen differentially displayed cDNA bands were isolated, cloned and verified by RT-PCR. The four mRNAs were increased, which included mRNAs encoding ***heat*** ***shock*** ***protein*** 27, fatty acid binding protein, apolipoprotein D and one novel gene. Six down-regulated clones were microtubule-associated protein IB, protein tyrosine phosphatase alpha, Kv1.2 channel, myelin protein SR13, medium-sized neurofilament protein, and one novel gene. Our results show that many differentially regulated genes after CCI may play a role in nerve degeneration and/or regeneration and provide a molecular framework for understanding the peripheral mechanism underlying neuropathic ***pain*** .
AB. . . differentially displayed cDNA bands were isolated, cloned and verified by RT-PCR. The four mRNAs were increased, which included mRNAs encoding ***heat*** ***shock*** ***protein*** 27, fatty acid binding protein, apolipoprotein D and one novel gene. Six down-regulated clones were microtubule-associated protein IB, protein tyrosine. . . play a role in nerve degeneration and/or regeneration and provide a molecular framework for understanding the peripheral mechanism underlying neuropathic ***pain*** .
IT . . .
nervous system; nerve: nervous system, degeneration, regeneration; peripheral nerve: nervous system, injury; sciatic nerve: nervous system, injury
IT Diseases
neuropathic ***pain*** : nervous system disease
Pain (MeSH)
IT Chemicals & Biochemicals
Kv1.2 channel; apolipoprotein D; cDNA [complementary DNA]; fatty acid binding protein; genes: differential expression; ***heat*** ***shock*** ***protein*** 27; mRNA [messenger RNA]; medium-sized neurofilament protein; microtubule-associated protein IB; myelin protein SR13; protein tyrosine phosphatase-alpha

L6 ANSWER 17 OF 41 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 2000:385077 BIOSIS <<LOGINID::20080330>>
DN PREV200000385077
TI Unstable angina activates myocardial ***heat*** ***shock*** ***protein*** 72, endothelial nitric oxide synthase, and transcription factors NFkappaB and AP-1.
AU Valen, Guro [Reprint author]; Hansson, Goran K.; Dumitrescu, Alexandra; Vaage, Jarle
CS Crafoord Laboratory of Experimental Surgery, Karolinska Hospital, S-171 76, Stockholm, Sweden
SO Cardiovascular Research, (July, 2000) Vol. 47, No. 1, pp. 49-56. print.
CODEN: CVREAU. ISSN: 0008-6363.
DT Article
LA English
ED Entered STN: 6 Sep 2000

Last Updated on STN: 8 Jan 2002

AB Objective: Unstable angina may improve the clinical outcome of acute myocardial infarction, but increases the morbidity and mortality of open heart surgery. We hypothetized that unstable angina influences the myocardium, and investigated the expression of the inducible ***heat*** ***shock*** ***protein*** 72 (HSP72), constitutive HSP73, and endothelial nitric oxide synthase (eNOS), and activation of the transcription factors NFkappaB and AP-1 in cardiac tissue. Methods: Biopsies were taken from the right atrium of 15 patients with unstable and 15 with stable angina undergoing coronary artery bypass grafting. Immunoblotting with monoclonal antibodies against HSP72, HSP73, and eNOS were performed on protein extracts, while nuclear proteins were assessed by electromobility shift assay. Results: When calculating the optical density of the bands, patients with unstable angina had more than twice as much HSP72 and eNOS as stable patients ($P < 0.005$), while HSP73 was similar in both groups. Nuclear translocation of NFkappaB and AP-1 was found in patients with anginal ***pain*** shortly before surgery, but not in stable patients or in patients without symptoms for 4 days or more prior to surgery. Conclusions: HSP72 and eNOS, which may be associated with cardioprotection in ischemic preconditioning, are increased in atrial tissue of patients with unstable angina. Activation of NFkappaB and AP-1, which regulate a battery of inflammatory genes, was found in hearts of unstable patients. NFkappaB activation may induce a myocardial proinflammatory state, possibly making the unstable myocardium more susceptible to the inflammation induced by open heart surgery.

TI Unstable angina activates myocardial ***heat*** ***shock*** ***protein*** 72, endothelial nitric oxide synthase, and transcription factors NFkappaB and AP-1.

AB. . . mortality of open heart surgery. We hypothetized that unstable angina influences the myocardium, and investigated the expression of the inducible ***heat*** ***shock*** ***protein*** 72 (HSP72), constitutive HSP73, and endothelial nitric oxide synthase (eNOS), and activation of the transcription factors NFkappaB and AP-1 in. . . 0.005), while HSP73 was similar in both groups. Nuclear translocation of NFkappaB and AP-1 was found in patients with anginal ***pain*** shortly before surgery, but not in stable patients or in patients without symptoms for 4 days or more prior to. . .

IT . . .
unstable angina: heart disease, vascular disease
Angina, Unstable (MeSH)

IT Chemicals & Biochemicals
AP-1: transcription factor; NF-kappaB [nuclear factor-kappaB]: transcription factor; ***heat*** ***shock*** ***protein*** 72: myocardial; nitric oxide synthase: endothelial

L6 ANSWER 18 OF 41 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AN 1999:79766 BIOSIS <<LOGINID::20080330>>

DN PREV199900079766

TI Spinal cord expression of ***heat*** ***shock*** ***protein*** (HSP27) in a rat model of neuropathic ***pain*** following tight ligation of L5 and L6 dorsal roots.

AU Allen, G. V. [Reprint author]; Esser, M. J.; Plumier, J.-C. L.; Sawynok, J.

CS Dep. Anatomy, Dalhousie Univ., Halifax, NS B3H 4H6, Canada

SO Society for Neuroscience Abstracts, (1998) Vol. 24, No. 1-2, pp. 1387. print.

Meeting Info.: 28th Annual Meeting of the Society for Neuroscience, Part 2. Los Angeles, California, USA. November 7-12, 1998.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LA English
ED Entered STN: 1 Mar 1999
Last Updated on STN: 1 Mar 1999
TI Spinal cord expression of ***heat*** ***shock*** ***protein*** (HSP27) in a rat model of neuropathic ***pain*** following tight ligation of L5 and L6 dorsal roots.
IT . . .
Systems of Organisms
spinal cord: nervous system; L5 dorsal root: nervous system; L6 dorsal root: nervous system
IT Diseases
neuropathic ***pain***
Pain (MeSH)
IT Diseases
peripheral nerve injury, nervous system
IT Chemicals & Biochemicals
heat ***shock*** ***protein*** 27: expression

L6 ANSWER 19 OF 41 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 1999:70553 BIOSIS <<LOGINID::20080330>>
DN PREV199900070553
TI Function of ***heat*** ***shock*** ***protein*** (HSP27) in neuropathic ***pain*** : Identification of new brain-specific HSP27-interacting proteins.
AU Plumier, J.-C. L. [Reprint author]; Esser, M. J.; Allen, G. V.; Sawynok, J.; Landry, J. [Reprint author]
CS Cancer Res. Cent., Laval Univ., Laval, PQ G1R 2J6, Canada
SO Society for Neuroscience Abstracts, (1998) Vol. 24, No. 1-2, pp. 1803. print.
Meeting Info.: 28th Annual Meeting of the Society for Neuroscience, Part 2. Los Angeles, California, USA. November 7-12, 1998. Society for Neuroscience.
ISSN: 0190-5295.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LA English
ED Entered STN: 1 Mar 1999
Last Updated on STN: 1 Mar 1999
TI Function of ***heat*** ***shock*** ***protein*** (HSP27) in neuropathic ***pain*** : Identification of new brain-specific HSP27-interacting proteins.
IT . . .
(Cell Biology); Nervous System (Neural Coordination)
IT Parts, Structures, & Systems of Organisms
glia: nervous system; neuron: nervous system; neuropathic ***pain***, nervous system disease
IT Chemicals & Biochemicals
brain-specific HSP27-interacting proteins [brain-specific ***heat*** ***shock*** ***protein*** 27-interacting proteins]; HSP27 [

heat ***shock*** ***protein*** 27]

L6 ANSWER 20 OF 41 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 1995:171547 BIOSIS <<LOGINID::20080330>>
DN PREV199598185847
TI Antimutagenic effect of stress adaptation.
AU Meerson, F. Z.; Kulakova, A. V.; Saltykova, V. A.
CS Inst. Gen. Pathol. Pathophysiol., Russ. Acad. Med. Sci., Moscow, Russia
SO Byulleten' Eksperimental'noi Biologii i Meditsiny, (1993) Vol. 115, No. 9, pp. 292-295.
CODEN: BEBMAE. ISSN: 0365-9615.
DT Article
LA Russian
ED Entered STN: 26 Apr 1995
Last Updated on STN: 27 Apr 1995
AB C57BL mice were adapted to moderate periodic hypoxia and repeated electric ***pain*** stresses of limited intensity. These animals adapted to each of the factors and control animals were given the potent mutagen, free radical oxidation activator dioxidine in a single dose of 300 mg/kg. Dioxidine administered to unadapted animals resulted in chromosomal aberrations in 11% of stem bone marrow cells mainly due to the appearance of single and multiple chromosomes. Preadaptation to stress decreased the number of these dioxidine-induced chromosomal aberrations nearly twice. Adaptation to periodic hypoxia had no defensive action. As previously shown,. adaptation to repeated stresses leads to the accumulation of ***heat*** - ***shock*** ***proteins*** (HSP) in the cellular nuclei of animals and prevents the degradation of isolated nuclei when single-chain DNA is added. Adaptation to hypoxia does not cause nuclear accumulation of HSP or prevents their degradation when unicellular DNA is supplemented. This suggests that the antimutagenic effect of stress adaptation is likely to be accounted for by the stabilizing action of HSP.
AB C57BL mice were adapted to moderate periodic hypoxia and repeated electric ***pain*** stresses of limited intensity. These animals adapted to each of the factors and control animals were given the potent mutagen,. . . Adaptation to periodic hypoxia had no defensive action. As previously shown,. adaptation to repeated stresses leads to the accumulation of ***heat*** - ***shock*** ***proteins*** (HSP) in the cellular nuclei of animals and prevents the degradation of isolated nuclei when single-chain DNA is added. Adaptation. . .
IT Miscellaneous Descriptors
BONE MARROW; CHROMOSOME ABERRATION; DIOXIDINE; DNA; ***HEAT*** ***SHOCK*** ***PROTEIN*** ; HYPOXIA; MUTAGENIC EFFECT; ***PAIN***

L6 ANSWER 21 OF 41 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN
AN 1995:22312 BIOSIS <<LOGINID::20080330>>
DN PREV199598036612
TI Arthritis and foodborne bacteria.
AU Smith, James L.
CS Eastern Regional Res. Cent., U.S. Dep. Agric., Agric. Res. Serv., Philadelphia, PA 19118, USA
SO Journal of Food Protection, (1994) Vol. 57, No. 10, pp. 935-941.
CODEN: JFPRDR. ISSN: 0362-028X.

DT Article
General Review; (Literature Review)
LA English
ED Entered STN: 11 Jan 1995
Last Updated on STN: 11 Jan 1995
AB Diarrheic episodes caused by the foodborne pathogens Campylobacter, Salmonella, Shigella or Yersinia may lead to a sterile arthritis such as reactive arthritis, Reiter' s syndrome or ankylosing spondylitis. Reiter's syndrome and reactive arthritis have been shown to be sequelae in a few well-studied bacterial food poisoning outbreaks. Reactive arthritis, Reiter's syndrome and ankylosing spondylitis show strong familial association related to the gene for HLA-B27 (HLA = human leucocyte antigen) antigen. Why HLA-B27-positive individuals are more susceptible to arthritis is not known, but molecular mimicry between the HLA-B27 antigen and antigens of triggering bacteria has been demonstrated and this mimicry has been proposed as a mechanism involved in etiology of the arthritides. Antigens from bacteria that triggered the arthritis are present in arthritic joints but bacterial cells are not found. Antibodies and T-cells specific for the triggering bacteria have been demonstrated in arthritic patients. T-cells present in synovial joints respond specifically to the particular arthritic triggering pathogen. The cells that respond to bacterial antigens belong to the T-cell subset T-H1 that secrete a limited number of cytokines but it is not known if cytokines are involved in arthritis. A few studies have demonstrated that T-cells from the joints of arthritic patients respond to both bacterial and human ***heat*** ***shock*** ***proteins*** indicating that autoimmunity may be involved in causation of arthritis. While only about 2% of a population exposed to a triggering infection will acquire arthritis, these individuals undergo ***pain*** and suffering as well as economic hardships as a result of their disease.
AB. . . arthritis. A few studies have demonstrated that T-cells from the joints of arthritic patients respond to both bacterial and human ***heat*** ***shock*** ***proteins*** indicating that autoimmunity may be involved in causation of arthritis. While only about 2% of a population exposed to a triggering infection will acquire arthritis, these individuals undergo ***pain*** and suffering as well as economic hardships as a result of their disease.

L6 ANSWER 22 OF 41 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2007:1453531 CAPLUS <<LOGINID::20080330>>
DN 148:305746
TI ***Heat*** ***shock*** ***protein*** 70 level of synovial fluid in rheumatoid arthritis versus osteoarthritis: a comparative study
AU Gharibdoost, F.; Samadi, F.; Taghipoor, R.; Akbarian, M.; Shahram, F.; Nadji, A.; Jamshidi, A. R.; Davatchi, F.
CS Rheumatology Research Center, Tehran University of Medical Sciences, Iran
SO Majalah Danishkadah Pezeshki (2007), 65(7), 28-31
CODEN: MDPACS
PB Tehran University of Medical Sciences
DT Journal
LA Persian
AB Background: ***Heat*** - ***shock*** ***proteins*** are part of a strictly controlled biol. system that allows organisms to respond to environmental stresses. Different proinflammatory cytokines are present in the synovial tissue of rheumatoid arthritis patients. Such tissues respond to stress and induce ***heat*** - ***shock*** ***proteins*** . In addn., synovial cells are exposed to mech. stress

caused by joint motion. The effects of mech. stress on the metab. of the synovial cells may be substantial, even pathogenic. ***Heat*** - ***shock*** ***proteins*** are often implicated in the pathogenesis of rheumatoid arthritis. Here, we compare the levels of ***heat*** - ***shock*** ***protein*** 70 from the synovial fluid of rheumatoid arthritis and osteoarthritis patients. Methods: Synovial fluid samples from 34 rheumatoid arthritis patients and 34 osteoarthritis patients were analyzed for ***heat*** - ***shock*** ***protein*** 70 by an ELISA method. Statistical anal. was performed using independent T-test and one-way ANOVA. Differences were considered statistically significant at p<0.05. Results: The mean value of synovial fluid ***heat*** - ***shock*** ***protein*** 70 levels in rheumatoid arthritis patients was 156.30.+-.128.51 and that of osteoarthritis patients was 14.98.+-.11.58. The differences were statistically significant at p<0.0001. For seven rheumatoid arthritis patients suffering from mech. knee ***pain*** , synovial fluid anal. revealed non-inflammatory effusion. The mean value of synovial fluid ***heat*** - ***shock*** ***protein*** 70 level in inflammatory synovial fluid of rheumatoid arthritis patients was significantly higher at 191.+-.121.73 and that of non-inflammatory synovial fluid from rheumatoid arthritis patients was 21.93.+-.10.06 (p<0.05). Conclusion: The level of ***heat*** ***shock*** ***protein*** 70 is higher in inflammatory arthritis than in non-inflammatory arthritis. Considering that patients with rheumatoid arthritis are known to have a hypertrophic synovial-lining layer, and that ***heat*** - ***shock*** ***protein*** 70 is known to protect cells against a variety of toxic conditions as well as apoptotic death, further research is needed to det. if ***heat*** - ***shock*** ***protein*** 70 induction is a sign of significant changes in the cellular and tissue metab. or is actively participating in the pathogenesis of rheumatoid arthritis.

TI ***Heat*** ***shock*** ***protein*** 70 level of synovial fluid in rheumatoid arthritis versus osteoarthritis: a comparative study

AB Background: ***Heat*** - ***shock*** ***proteins*** are part of a strictly controlled biol. system that allows organisms to respond to environmental stresses. Different proinflammatory cytokines are present in the synovial tissue of rheumatoid arthritis patients. Such tissues respond to stress and induce ***heat*** - ***shock*** ***proteins*** . In addn., synovial cells are exposed to mech. stress caused by joint motion. The effects of mech. stress on the metab. of the synovial cells may be substantial, even pathogenic. ***Heat*** - ***shock*** ***proteins*** are often implicated in the pathogenesis of rheumatoid arthritis. Here, we compare the levels of ***heat*** - ***shock*** ***protein*** 70 from the synovial fluid of rheumatoid arthritis and osteoarthritis patients. Methods: Synovial fluid samples from 34 rheumatoid arthritis patients and 34 osteoarthritis patients were analyzed for ***heat*** - ***shock*** ***protein*** 70 by an ELISA method. Statistical anal. was performed using independent T-test and one-way ANOVA. Differences were considered statistically significant at p<0.05. Results: The mean value of synovial fluid ***heat*** - ***shock*** ***protein*** 70 levels in rheumatoid arthritis patients was 156.30.+-.128.51 and that of osteoarthritis patients was 14.98.+-.11.58. The differences were statistically significant at p<0.0001. For seven rheumatoid arthritis patients suffering from mech. knee ***pain*** , synovial fluid anal. revealed non-inflammatory effusion. The mean value of synovial fluid ***heat*** - ***shock***

protein 70 level in inflammatory synovial fluid of rheumatoid arthritis patients was significantly higher at 191.+-.121.73 and that of non-inflammatory synovial fluid from rheumatoid arthritis patients was 21.93.+-.10.06 ($p<0.05$). Conclusion: The level of ***heat*** ***shock*** ***protein*** 70 is higher in inflammatory arthritis than in non-inflammatory arthritis. Considering that patients with rheumatoid arthritis are known to have a hypertrophic synovial-lining layer, and that ***heat*** - ***shock*** ***protein*** 70 is known to protect cells against a variety of toxic conditions as well as apoptotic death, further research is needed to det. if ***heat*** - ***shock*** ***protein*** 70 induction is a sign of significant changes in the cellular and tissue metab. or is actively participating in the. . .

IT ***Heat*** - ***shock*** ***proteins***
RL: BSU (Biological study, unclassified); DGN (Diagnostic use); BIOL (Biological study); USES (Uses)
(HSP 70; ***heat*** ***shock*** ***protein*** 70 level of synovial fluid in rheumatoid arthritis vs. osteoarthritis)

IT Apoptosis
Human
Osteoarthritis
Rheumatoid arthritis
Synovial fluid
(***heat*** ***shock*** ***protein*** 70 level of synovial fluid in rheumatoid arthritis vs. osteoarthritis)

L6 ANSWER 23 OF 41 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2007:538484 CAPLUS <<LOGINID::20080330>>
DN 146:516127
TI Poly (ADP-ribose) polymerase PARP inhibitors derived from C-terminal non-histone domain of macroH2A histone and its therapeutic uses
IN Hamiche, Ali
PA Centre National de la Recherche Scientifique - CNRS, Fr.
SO PCT Int. Appl., 105pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2007054814	A1	20070518	WO 2006-IB3224	20061114

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, GT, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRAI US 2005-736588P P 20051114

AB The present invention relates to new inhibitors of poly(adenosine 5'-diphospho-ribose) polymerase ["poly(ADP-ribose) polymerase" or "PARP", which is also sometimes called "PARS" for poly(ADP-ribose) synthetase].

The PARP inhibitors are derived from C-terminal non-histone domain of macroH2A histone. More particularly, the invention relates to the use of PARP inhibitors to prevent and/or treat tissue damage resulting from cell damage or death due to necrosis or apoptosis; neural tissue damage resulting from ischemia and reperfusion injury; neurol. disorders and neurodegenerative diseases; vascular stroke; cardiovascular disorders; other conditions and/or disorders such as age-related macular degeneration, AIDS and other immune senescence diseases, arthritis, atherosclerosis, cachexia, cancer, degenerative diseases of skeletal muscle involving replicative senescence, diabetes, head trauma, immune senescence, inflammatory bowel disorders, muscular dystrophy, osteoarthritis, osteoporosis, chronic and acute ***pain*** , renal failure, retinal ischemia, septic ***shock*** (such as endotoxic ***shock***), and skin aging. The PARP inhibitors are also useful in extending the lifespan and proliferative capacity of cells; altering gene expression of senescent cells; or radiosensitizing hypoxic tumor cells. MacroH2A1.1 interacted with PARP-1 through its C-terminal non-histone region. MacroH2A1.1 targeted specifically the hsp70 promoter. Heat ***shock*** induced macroH2A1.1 and PARP-1 displacement from the hsp70-1 promoter. Down regulation of macroH2A1.1 or PARP-1 delayed heat ***shock*** response. MacroH2A1.1 regulated PARP-1 enzymic activity. The sequences of human macrH2A1.1, macrH2A1.2, and macrH2A2 are provided.

RE.CNT 9 THERE ARE 9 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB . . . skeletal muscle involving replicative senescence, diabetes, head trauma, immune senescence, inflammatory bowel disorders, muscular dystrophy, osteoarthritis, osteoporosis, chronic and acute ***pain*** , renal failure, retinal ischemia, septic ***shock*** (such as endotoxic ***shock***), and skin aging. The PARP inhibitors are also useful in extending the lifespan and proliferative capacity of cells; altering gene. . . radiosensitizing hypoxic tumor cells. MacroH2A1.1 interacted with PARP-1 through its C-terminal non-histone region. MacroH2A1.1 targeted specifically the hsp70 promoter. Heat ***shock*** induced macroH2A1.1 and PARP-1 displacement from the hsp70-1 promoter. Down regulation of macroH2A1.1 or PARP-1 delayed heat ***shock*** response. MacroH2A1.1 regulated PARP-1 enzymic activity. The sequences of human macrH2A1.1, macrH2A1.2, and macrH2A2 are provided.

IT ***Heat*** - ***shock*** ***proteins***
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(HSP 70, promoter, macroH2A targeting; poly (ADP-ribose) polymerase PARP inhibitors derived from C-terminal non-histone domain of macroH2A histone and its therapeutic uses)

IT ***Pain***
(acute, treatment of; poly (ADP-ribose) polymerase PARP inhibitors derived from C-terminal non-histone domain of macroH2A histone and its therapeutic uses)

IT ***Pain***
(chronic, treatment of; poly (ADP-ribose) polymerase PARP inhibitors derived from C-terminal non-histone domain of macroH2A histone and its therapeutic uses)

L6 ANSWER 24 OF 41 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2007:289359 CAPLUS <<LOGINID::20080330>>
DN 147:86506
TI Phase I and pharmacodynamic study of 17-(allylamino)-17-demethoxygeldanamycin in adult patients with refractory advanced cancers

AU Ramanathan, Ramesh K.; Egorin, Merrill J.; Eiseman, Julie L.; Ramalingam, Suresh; Friedland, David; Agarwala, Sanjiv S.; Ivy, S. Percy; Potter, Douglas M.; Chatta, Gurkamal; Zuhowski, Eleanor G.; Stoller, Ronald G.; Naret, Cynthia; Guo, Jianxia; Belani, Chandra P.

CS Division of Hematology/Oncology, Department of Medicine, Molecular Therapeutics/Drug Discovery Program, University of Pittsburg Graduate School of Public Health and Biostatistics Facility, University of Pittsburgh Cancer Institute, University of Pittsburgh School of Medicine, Pittsburgh, PA, USA

SO Clinical Cancer Research (2007), 13(6), 1769-1774
CODEN: CCREF4; ISSN: 1078-0432

PB American Association for Cancer Research

DT Journal

LA English

AB The primary objective was to establish the dose-limiting toxicity (DLT) and recommended phase II dose of 17-(allylamino)-17-demethoxygeldanamycin (17AAG) given twice a week. Escalating doses of 17AAG were given i.v. to cohorts of three to six patients. Dose levels for schedule A (twice weekly x 3 wk, every 4 wk) were 100, 125, 150, 175, and 200 mg/m2 and for schedule B (twice weekly x 2 wk, every 3 wk) were 150, 200, and 250 mg/m2. Peripheral blood mononuclear cells (PBMC) were collected for assessment of ***heat*** ***shock*** ***protein*** (HSP) 90 and HSP90 client proteins. Forty-four patients were enrolled, 32 on schedule A and 12 on schedule B. On schedule A at 200 mg/m2, DLTs were seen in two of six patients (one grade 3 thrombocytopenia and one grade 3 abdominal ***pain***). On schedule B, both patients treated at 250 mg/m2 developed DLT (grade 3 headache with nausea/vomiting). Grade 3/4 toxicities seen in >5% of patients were reversible elevations of liver enzymes (47%), nausea (9%), vomiting (9%), and headache (5%). No objective tumor responses were obsd. The only consistent change in PBMC proteins monitored was a 0.8- to 30-fold increase in HSP70 concns., but these were not dose dependent. The increase in PBMC HSP70 persisted throughout the entire cycle of treatment but returned to baseline between last 17AAG dose of cycle 1 and first 17AAG dose of cycle 2. The recommended phase II doses of 17AAG are 175 to 200 mg/m2 when given twice a week and consistently cause elevations in PBMC HSP70.

RE.CNT 22 THERE ARE 22 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB . . . wk, every 3 wk) were 150, 200, and 250 mg/m2. Peripheral blood mononuclear cells (PBMC) were collected for assessment of ***heat*** ***shock*** ***protein*** (HSP) 90 and HSP90 client proteins. Forty-four patients were enrolled, 32 on schedule A and 12 on schedule B. On. . . at 200 mg/m2, DLTs were seen in two of six patients (one grade 3 thrombocytopenia and one grade 3 abdominal ***pain***). On schedule B, both patients treated at 250 mg/m2 developed DLT (grade 3 headache with nausea/vomiting). Grade 3/4 toxicities seen. . .

ST pharmacodynamics allylamino demethoxygeldanamycin anticancer ***heat*** ***shock*** ***protein*** cancer sarcoma

IT Mononuclear cell (leukocyte)
(17-(allylamino)-17-demethoxygeldanamycin at recommended phase II dose of 175 to 200 mg/m2 elevated ***heat*** ***shock*** ***protein*** -70 of peripheral blood mononuclear cell in patient with
refractory advanced cancer)

IT Esophagus, neoplasm
(17-(allylamino)-17-demethoxygeldanamycin at recommended phase II dose of 175 to 200 mg/m2 elevated ***heat*** ***shock***

protein -70 of peripheral blood mononuclear cell while was
effective and tolerated in patient with esophageal cancer)
IT Head and Neck, neoplasm
(17-(allylamino)-17-demethoxygeldanamycin at recommended phase II dose
of 175 to 200 mg/m2 elevated ***heat*** ***shock***
protein -70 of peripheral blood mononuclear cell while was
effective and tolerated in patient with head and neck cancer)
IT Pancreas, neoplasm
(17-(allylamino)-17-demethoxygeldanamycin at recommended phase II dose
of 175 to 200 mg/m2 elevated ***heat*** ***shock***
protein -70 of peripheral blood mononuclear cell while was
effective and tolerated in patient with pancreas cancer)
IT Prostate gland, neoplasm
(17-(allylamino)-17-demethoxygeldanamycin at recommended phase II dose
of 175 to 200 mg/m2 elevated ***heat*** ***shock***
protein -70 of peripheral blood mononuclear cell while was
effective and tolerated in patient with prostate cancer)
IT Antitumor agents
Drug toxicity
Human
Pharmacodynamics
(17-(allylamino)-17-demethoxygeldanamycin at recommended phase II dose
of 175 to 200 mg/m2 elevated ***heat*** ***shock***
protein -70 of peripheral blood mononuclear cell while was
effective and tolerated in patient with refractory advanced cancer)
IT Sarcoma
(17-(allylamino)-17-demethoxygeldanamycin at recommended phase II dose
of 175 to 200 mg/m2 elevated ***heat*** ***shock***
protein -70 of peripheral blood mononuclear cell while was
effective and tolerated in patient with sarcoma)
IT ***Heat*** - ***shock*** ***proteins***
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(HSP 70; 17-(allylamino)-17-demethoxygeldanamycin at recommended phase
II dose of 175 to 200 mg/m2 elevated ***heat*** ***shock***
protein -70 of peripheral blood mononuclear cell in patient
with
refractory advanced cancer)
IT ***Heat*** - ***shock*** ***proteins***
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(HSP 90; 17-(allylamino)-17-demethoxygeldanamycin at recommended phase
II dose of 175 to 200 mg/m2 did not change ***heat*** ***shock***
protein -90 in patient with refractory advanced cancer)
IT Intestine, neoplasm
(colorectal; 17-(allylamino)-17-demethoxygeldanamycin at recommended
phase II dose of 175 to 200 mg/m2 elevated ***heat*** ***shock***
protein -70 of peripheral blood mononuclear cell while was
effective and tolerated in patient with colorectal cancer)
IT Neoplasm
(head and neck; 17-(allylamino)-17-demethoxygeldanamycin at recommended
phase II dose of 175 to 200 mg/m2 elevated ***heat*** ***shock***
protein -70 of peripheral blood mononuclear cell while was
effective and tolerated in patient with head and neck cancer)
IT Lung, neoplasm
(non-small-cell carcinoma; 17-(allylamino)-17-demethoxygeldanamycin at
recommended phase II dose of 175 to 200 mg/m2 elevated ***heat***
shock ***protein*** -70 of peripheral blood mononuclear
cell

while was effective and tolerated in patient with non-small cell lung cancer)

IT Carcinoma
(pulmonary non-small-cell; 17-(allylamino)-17-demethoxygeldanamycin at recommended phase II dose of 175 to 200 mg/m2 elevated ***heat*** ***shock*** ***protein*** -70 of peripheral blood mononuclear cell
while was effective and tolerated in patient with non-small cell lung cancer)

IT 75747-14-7, 17-(Allylamino)-17-demethoxygeldanamycin
RL: ADV (Adverse effect, including toxicity); PAC (Pharmacological activity); PKT (Pharmacokinetics); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(17-(allylamino)-17-demethoxygeldanamycin at recommended phase II dose of 175 to 200 mg/m2 elevated ***heat*** ***shock*** ***protein*** -70 of peripheral blood mononuclear cell while was effective and tolerated in patient with refractory advanced cancer)

L6 ANSWER 25 OF 41 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2007:223069 CAPLUS <<LOGINID::20080330>>
TI Muscle ***pain*** suppressant and functional food for muscle ***pain*** suppression
IN Yoshikawa, Toshikazu; Naito, Yuji; Aoi, Wataru; Takano, Toshiaki; Masuyama, Akihiro; Nakamura, Teppei
PA Calpis Co., Ltd., Japan
SO PCT Int. Appl.
CODEN: PIXXD2
DT Patent
LA Japanese
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2007023896	A1	20070301	WO 2006-JP316610	20060824

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HN, HR, HU, ID, IL, IN, IS, JP, KE, KG, KM, KN, KP, KR, KZ, LA, LC, LK, LR, LS, LT, LU, LV, LY, MA, MD, MG, MK, MN, MW, MX, MY, MZ, NA, NG, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RS, RU, SC, SD, SE, SG, SK, SL, SM, SV, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, ZA, ZM, ZW
RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, LV, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRAI JP 2005-246312 A 20050826

AB A muscle ***pain*** suppressant, or functional food, that can be continuously taken on a daily basis, excelling in safety, and that can relieve or prevent the onset of muscle ***pain*** , being especially effective in the suppression of delayed onset muscle soreness. As an active ingredient, there is contained a fermented milk preferably exhibiting at least one of antioxidant enzyme developing potency, ***heat*** ***shock*** ***protein*** developing potency, and neutrotaxis and tissue infiltration inhibiting potency, which fermented milk is obtained by fermenting milk by the use of microbial cells containing Lactobacillus helveticus.

TI Muscle ***pain*** suppressant and functional food for muscle

pain suppression

AB A muscle ***pain*** suppressant, or functional food, that can be continuously taken on a daily basis, excelling in safety, and that can relieve or prevent the onset of muscle ***pain*** , being especially effective in the suppression of delayed onset muscle soreness. As an active ingredient, there is contained a fermented milk preferably exhibiting at least one of antioxidant enzyme developing potency, ***heat*** ***shock*** ***protein*** developing potency, and neutrotaxis and tissue infiltration inhibiting potency, which fermented milk is obtained by fermenting milk by the use. . .

L6 ANSWER 26 OF 41 CAPLUS COPYRIGHT 2008 ACS on STN

AN 2005:1097743 CAPLUS <<LOGINID::20080330>>

DN 144:462396

TI Cytotoxicity and expression of c-fos, HSP70, and GADD45/153 proteins in human liver carcinoma (HepG2) cells exposed to dinitrotoluenes

AU Glass, Konsuela Y.; Newsome, Cecilia R.; Tchounwou, Paul B.

CS Molecular Toxicology Research Laboratory, NIH-Center for Environmental Health, School of Science and Technology, Jackson State University, Jackson, MS, 39217, USA

SO International Journal of Environmental Research and Public Health (2005), 2(2), 355-361
CODEN: IJERGQ; ISSN: 1660-4601
URL: http://www.mdpi.org/ijerph/papers2/ijerph2005020022.pdf

PB Molecular Diversity Preservation International

DT Journal; (online computer file)

LA English

AB Dinitrotoluenes (DNTs) are byproducts of the explosive trinitrotoluene (TNT), and exist as a mixt. of 2 to 6 isomers, with 2,4-DNT and 2,6-DNT being the most significant. The main route of human exposure at ammunition facilities is inhalation. The primary targets of DNTs toxicity are the hematopoietic system, cardiovascular system, nervous system and reproductive system. In factory workers, exposure to DNTs has been linked to many adverse health effects, including: cyanosis, vertigo, headache, metallic taste, dyspnea, weakness and lassitude, loss of appetite, nausea, and vomiting. Other symptoms including ***pain*** or parasthesia in extremities, abdominal discomfort, tremors, paralysis, chest ***pain*** , and unconsciousness have been documented. An assocn. between DNTs exposure and increased risk of hepatocellular carcinomas and s.c. tumors in rats, as well as renal tumors in mice, has been established. This research was therefore designed targeting the liver to assess the cellular and mol. responses of human liver carcinoma cells following exposure to 2,4-DNT and 2,6-DNT. Cytotoxicity was evaluated using the MTT assay. Upon 48 h of exposure, LC50 values of 245 .+-. 14.72 .mu.g/mL, and 300 .+-. 5.92 .mu.g/mL were recorded for 2,6-DNT and 2,4-DNT resp., indicating that both DNTs are moderately toxic, and 2,6-DNT is slightly more toxic to HepG2 cells than 2,4-DNT. A dose response relationship was recorded with respect to the cytotoxicity of both DNTs. Western blot anal. resulted in a significant expression ($p < 0.05$) of the 70-kDa ***heat*** ***shock*** ***protein*** in 2,6-DNT-treated cells compared to the control cells and at the 200 .mu.g/mL dose for 2,4-DNT. A statistically significant expression in c-fos was also obsd. at the 200 and 250 .mu.g/mL treatment level for 2,4- and 2,6-DNT, resp. However, no statistically significant expression of this protooncogene-related protein was obsd. at the doses of 0, 100, or 300 .mu.g/mL or within the dose range of 0-200 .mu.g/mL for 2,6-DNT. The 45-kDa growth arrest and damage protein was significantly expressed at the dose range of 200 - 250 .mu.g/mL for

2,6-DNT and at the dose range of 200 - 400 .mu.g/mL for 2,4-DNT. Expression of 153-kDa growth arrest and DNA damage protein was significant at the 100, 200, and 250 .mu.g/mL doses for 2,6-DNT and at the 200 .mu.g/mL dose for 2,4-DNT. Overall, these results indicate the potential of DNTs to induce cytotoxic, proteotoxic (HSP70), and genotoxic (GADD45/153) effects, as well as oxidative stress and pro-inflammatory reactions (c-fos).

RE.CNT 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB . . . effects, including: cyanosis, vertigo, headache, metallic taste, dyspnea, weakness and lassitude, loss of appetite, nausea, and vomiting. Other symptoms including ***pain*** or parasthesia in extremities, abdominal discomfort, tremors, paralysis, chest ***pain*** , and unconsciousness have been documented. An assocn. between DNTs exposure and increased risk of hepatocellular carcinomas and s.c. tumors in. . . to the cytotoxicity of both DNTs. Western blot anal. resulted in a significant expression ($p < 0.05$) of the 70-kDa ***heat*** ***shock*** ***protein*** in 2,6-DNT-treated cells compared to the control cells and at the 200 .mu.g/mL dose for 2,4-DNT. A statistically significant expression. . .

L6 ANSWER 27 OF 41 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2004:1020566 CAPLUS <<LOGINID::20080330>>
DN 142:53699
TI Evaluation of using HSP72 in lymphocytes as a prognostic criterion in myocardial infarction
AU Drapkina, O. M.; Zadorozhnaya, O. O.; Malyshev, I. Yu.; Ivashkin, V. T.
CS Klin. Propedevtiki Vnutren. Bolezn., Gastroenterol. i Gepatol. im. Akad. V. KH. Vasilenko, MMA im. I. M. Sechenova, Russia
SO Molekulyarnaya Meditsina (Moscow, Russian Federation) (2004), (2), 49-60 CODEN: MMMRCP; ISSN: 1728-2918
PB Izdatel'stvo Meditsina
DT Journal
LA Russian
AB Myocardial resistance to damage depends on activity of intracellular defense systems including the family of ***heat*** ***shock*** ***proteins*** (HSP72) or stress proteins. We studied HSP72 synthesis in patients with transmural myocardial infarction (MI), compared changes in HSP72 synthesis with clin. picture of Ml and tried possibility of HSP72 application as a prognostic criterion in the course and outcome of Ml. A total of 25 patients (23 males and 2 females) with transmural Ml (Q-infarction) were examd. The diagnosis was made basing on the ***pain*** syndrome, ECG findings and high activity of serum enzymes. Basal and inducable levels of HSP72 on Ml day 1 were measured in the lymphocytes of peripheral blood. Patients with different stress responses differ also by clin. picture of the disease. The level of HSP72 can be used as an addnl. prognostic criterion of Ml course.
AB Myocardial resistance to damage depends on activity of intracellular defense systems including the family of ***heat*** ***shock*** ***proteins*** (HSP72) or stress proteins. We studied HSP72 synthesis in patients with transmural myocardial infarction (MI), compared changes in HSP72 synthesis. . . 25 patients (23 males and 2 females) with transmural Ml (Q-infarction) were examd. The diagnosis was made basing on the ***pain*** syndrome, ECG findings and high activity of serum enzymes. Basal and inducable levels of HSP72 on Ml day 1 were. . .
IT ***Heat*** - ***shock*** ***proteins***
RL: BSU (Biological study, unclassified); DGN (Diagnostic use); BIOL

(Biological study); USES (Uses)
(HSP 72; evaluation of using HSP72 in lymphocytes as prognostic criterion in myocardial infarction)

L6 ANSWER 28 OF 41 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2004:119531 CAPLUS <<LOGINID::20080330>>
DN 140:175161
TI Heat ***shock*** polypeptides as ***pain*** relief agents
IN Coates, Anthony Robert Milnes
PA Helperby Therapeutics Limited, UK
SO Brit. UK Pat. Appl., 39 pp.
CODEN: BAXXDU
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	GB 2391477	A	20040211	GB 2003-25782	20031105
	GB 2391477	B	20041222		
	CA 2503964	A1	20040521	CA 2003-2503964	20031105
	WO 2004041304	A2	20040521	WO 2003-GB4774	20031105
	WO 2004041304	A3	20040729		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW				
	RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2003279454	A1	20040607	AU 2003-279454	20031105
	EP 1562625	A2	20050817	EP 2003-772402	20031105
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
	BR 2003016053	A	20050920	BR 2003-16053	20031105
	CN 1735427	A	20060215	CN 2003-80108562	20031105
	JP 2006514001	T	20060427	JP 2004-549345	20031105
	NO 2005002216	A	20050805	NO 2005-2216	20050509
	US 2006252681	A1	20061109	US 2006-534054	20060322
PRAI	GB 2002-26105	A	20021108		
	WO 2003-GB4774	W	20031105		

AB The present invention concerns the use of a heat ***shock*** polypeptide and/or an encoding nucleic acid sequence in the manuf. of a medicament for use in the relief of ***pain*** . In particular the invention concerns the use of chaperonin. The invention further provides methods of relieving ***pain*** using medicaments contg. the heat ***shock*** polypeptides.
RE.CNT 2 THERE ARE 2 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
TI Heat ***shock*** polypeptides as ***pain*** relief agents
AB The present invention concerns the use of a heat ***shock*** polypeptide and/or an encoding nucleic acid sequence in the manuf. of a medicament for use in the relief of ***pain*** . In particular the invention concerns the use of chaperonin. The invention further provides methods of relieving ***pain*** using medicaments contg. the heat

```
       ***shock***   polypeptides.
ST     ***pain***   relief heat shock polypeptides nucleic acid; chaperonin
       ***pain***   relief nucleic acid
IT   Disease, animal
        (back   ***pain***  ;   ***heat***     ***shock***     ***proteins***
        that are chaperonins as   ***pain***   relief agents and their encoding
        nucleic acids in relation to use with other analgesics)
IT   Body, anatomical
        (back, disease,   ***pain***  ;   ***heat***     ***shock***
          ***proteins***   that are chaperonins as   ***pain***   relief agents
        and their encoding nucleic acids in relation to use with other
        analgesics)
IT     ***Pain***
        (back;   ***heat***     ***shock***     ***proteins***   that are
        chaperonins as   ***pain***   relief agents and their encoding nucleic
        acids in relation to use with other analgesics)
IT   Drug delivery systems
        (carriers;   ***heat***     ***shock***     ***proteins***   that are
        chaperonins as   ***pain***   relief agents and their encoding nucleic
        acids in relation to use with other analgesics)
IT     ***Pain***
        (dental;   ***heat***     ***shock***     ***proteins***   that are
        chaperonins as   ***pain***   relief agents and their encoding nucleic
        acids in relation to use with other analgesics)
IT   Drug delivery systems
        (diluents;   ***heat***     ***shock***     ***proteins***   that are
        chaperonins as   ***pain***   relief agents and their encoding nucleic
        acids in relation to use with other analgesics)
IT   Viscera
        (disease,   ***pain***  ;   ***heat***     ***shock***
          ***proteins***   that are chaperonins as   ***pain***   relief agents
        and their encoding nucleic acids in relation to use with other
        analgesics)
IT   Ear, disease
        (earache;   ***heat***     ***shock***     ***proteins***   that are
        chaperonins as   ***pain***   relief agents and their encoding nucleic
        acids in relation to use with other analgesics)
IT   Drug delivery systems
        (excipients;   ***heat***     ***shock***     ***proteins***   that are
        chaperonins as   ***pain***   relief agents and their encoding nucleic
        acids in relation to use with other analgesics)
IT   Bone, disease
        (fracture,   ***pain***   from;   ***heat***     ***shock***
          ***proteins***   that are chaperonins as   ***pain***   relief agents
        and their encoding nucleic acids in relation to use with other
        analgesics)
IT   Nucleic acids
     RL: BSU (Biological study, unclassified); BUU (Biological use,
     unclassified); BIOL (Biological study); USES (Uses)
        (  ***heat***     ***shock***     ***protein***  -encoding;
          ***heat***     ***shock***     ***proteins***   that are chaperonins
as
          ***pain***   relief agents and their encoding nucleic acids in
relation
        to use with other analgesics)
IT   Eubacteria
     Mycobacterium
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Mycobacterium tuberculosis
(***heat*** ***shock*** ***proteins*** from; ***heat*** ***shock*** ***proteins*** that are chaperonins as ***pain*** relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Analgesics
Animals
Drug interactions
Headache
Human
Pain
(***heat*** ***shock*** ***proteins*** that are chaperonins as ***pain*** relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT ***Heat*** - ***shock*** ***proteins***
Molecular chaperones
Opioids
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(***heat*** ***shock*** ***proteins*** that are chaperonins as ***pain*** relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Drug delivery systems
(inhalants; ***heat*** ***shock*** ***proteins*** that are chaperonins as ***pain*** relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Drug delivery systems
(nasal; ***heat*** ***shock*** ***proteins*** that are chaperonins as ***pain*** relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Anti-inflammatory agents
(nonsteroidal; ***heat*** ***shock*** ***proteins*** that are chaperonins as ***pain*** relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Drug delivery systems
(ophthalmic; ***heat*** ***shock*** ***proteins*** that are chaperonins as ***pain*** relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Drug delivery systems
(oral; ***heat*** ***shock*** ***proteins*** that are chaperonins as ***pain*** relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Abscess
Arthritis
Burn
Gout
Infection
Inflammation
Menstruation
Neoplasm
Parturition
(***pain*** from; ***heat*** ***shock*** ***proteins*** that are chaperonins as ***pain*** relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT Tooth, disease
(***pain*** ; ***heat*** ***shock*** ***proteins*** that are chaperonins as ***pain*** relief agents and their encoding

nucleic acids in relation to use with other analgesics)
IT Drug delivery systems
(parenterals; ***heat*** ***shock*** ***proteins*** that are chaperonins as ***pain*** relief agents and their encoding nucleic acids in relation to use with other analgesics)
IT Surgery
(post-surgical ***pain*** from; ***heat*** ***shock*** ***proteins*** that are chaperonins as ***pain*** relief agents and their encoding nucleic acids in relation to use with other analgesics)
IT Kidney, disease
(renal tract ***pain*** ; ***heat*** ***shock*** ***proteins*** that are chaperonins as ***pain*** relief agents and their encoding nucleic acids in relation to use with other analgesics)
IT Animal tissue, disease
(soft, injury, ***pain*** from; ***heat*** ***shock*** ***proteins*** that are chaperonins as ***pain*** relief agents and their encoding nucleic acids in relation to use with other analgesics)
IT Drug delivery systems
(suppositories, vaginal; ***heat*** ***shock*** ***proteins*** that are chaperonins as ***pain*** relief agents and their encoding nucleic acids in relation to use with other analgesics)
IT Drug delivery systems
(suppositories; ***heat*** ***shock*** ***proteins*** that are chaperonins as ***pain*** relief agents and their encoding nucleic acids in relation to use with other analgesics)
IT Drug delivery systems
(topical; ***heat*** ***shock*** ***proteins*** that are chaperonins as ***pain*** relief agents and their encoding nucleic acids in relation to use with other analgesics)
IT Ligament
Tendon
(traumatic damage, ***pain*** from; ***heat*** ***shock*** ***proteins*** that are chaperonins as ***pain*** relief agents and their encoding nucleic acids in relation to use with other analgesics)
IT Disease, animal
(visceral ***pain*** ; ***heat*** ***shock*** ***proteins*** that are chaperonins as ***pain*** relief agents and their encoding nucleic acids in relation to use with other analgesics)
IT ***Pain***
(visceral; ***heat*** ***shock*** ***proteins*** that are chaperonins as ***pain*** relief agents and their encoding nucleic acids in relation to use with other analgesics)
IT 657078-79-0, Chaperonin 60.2 (Mycobacterium tuberculosis) 657078-80-3, Chaperonin 10 (Mycobacterium tuberculosis)
RL: BSU (Biological study, unclassified); PAC (Pharmacological activity); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(amino acid sequence; ***heat*** ***shock*** ***proteins*** that are chaperonins as ***pain*** relief agents and their encoding nucleic acids in relation to use with other analgesics)
IT 657085-31-9

RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(amino acid sequence; ***heat*** ***shock*** ***proteins*** that are chaperonins as ***pain*** relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT 50-78-2, Aspirin 103-90-2, Paracetamol 15687-27-1, Ibuprofen
RL: PAC (Pharmacological activity); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(***heat*** ***shock*** ***proteins*** that are chaperonins as ***pain*** relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT 329900-75-6, Cyclooxygenase 2
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(inhibitors; ***heat*** ***shock*** ***proteins*** that are chaperonins as ***pain*** relief agents and their encoding nucleic acids in relation to use with other analgesics)

IT 657078-76-7 657078-77-8 657078-78-9
RL: BSU (Biological study, unclassified); BUU (Biological use, unclassified); BIOL (Biological study); USES (Uses)
(nucleotide sequence; ***heat*** ***shock*** ***proteins*** that are chaperonins as ***pain*** relief agents and their encoding nucleic acids in relation to use with other analgesics)

L6 ANSWER 29 OF 41 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2002:987 CAPLUS <<LOGINID::20080330>>
DN 136:400402
TI Prognostic factors for radiographic damage in early rheumatoid arthritis: A multiparameter prospective study
AU Combe, B.; Dougados, M.; Goupille, P.; Cantagrel, A.; Eliaou, J. F.; Sibilia, J.; Meyer, O.; Sany, J.; Daures, J.-P.; Dubois, A.
CS Federation Rhumatologic, INSERMU 475, Centre Hospitalier Universitaire Montpellier, and INSERM U475, Montpellier, Fr.
SO Arthritis & Rheumatism (2001), 44(8), 1736-1743
CODEN: ARHEAW; ISSN: 0004-3591
PB Wiley-Liss, Inc.
DT Journal
LA English
AB Objective: To det. prognostic factors of radiol. damage and radiol. progression in early rheumatoid arthritis (RA). A cohort of 191 patients with RA whose disease duration was shorter than 1 yr were prospectively followed up for 3 yr. Radiol. scores (as detd. by Sharp's method, modified by van der Heijde) and radiol. progression were used as outcome measures. Numerous baseline clin., lab., genetic, and radiog. data were obtained. The change in the total radiol. score for the patients followed up over 3 yr was a mean .+-. SD increase of 6.1 .+-. 6.2. Radiol. progression was obsd. in 71 of the 172 patients for whom there were data at the end of the study. By univariate anal. with Fisher's exact test, radiol. scores and progression at followup were closely correlated with the baseline values of the erythrocyte sedimentation rate (ESR), C-reactive protein level, IgM and IgA rheumatoid factor positivity, antiperinuclear antibody positivity, radiol. scores, duration of morning stiffness, and RA-assocd. HLA-DRB1*04 genes. No correlation was demonstrated with sex, age, Disease Activity Score, swollen or tender joint counts, extraarticular manifestations, Health Assessment Questionnaire score, Ritchie Articular Index, patient's assessment of ***pain*** , positivity for anti- ***heat*** - ***shock*** ***protein*** 90-kd antibodies, anticalpastatin antibodies, anti-RA33

antibodies, antinuclear antibodies, YKL-40, or antikeratin antibodies, and HLA-DRB1*01 genes. The logistic regression anal. revealed that the only baseline values that were predictive of the 3-yr radiol. scores were IgM rheumatoid factor positivity, DRB1*04 genes, ***pain*** score, and total radiol. score. Progression of joint damage was predicted by the ESR, IgM rheumatoid factor positivity, DRB1*04 genes, and erosions score at baseline. Prognostic factors for radiog. damage in early RA were identified. A combination of these baseline values allowed us to draw up a predictive arithmetic score that could be used to predict radiol. damage at 3 yr and radiol. progression in individual patients.

RE.CNT 39 THERE ARE 39 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB . . . Disease Activity Score, swollen or tender joint counts, extraarticular manifestations, Health Assessment Questionnaire score, Ritchie Articular Index, patient's assessment of ***pain*** , positivity for anti- ***heat*** - ***shock*** ***protein*** 90-kd antibodies, anticalpastatin antibodies, anti-RA33 antibodies, antinuclear antibodies, YKL-40, or antikeratin antibodies, and HLA-DRB1*01 genes. The logistic regression anal. revealed. . . that the only baseline values that were predictive of the 3-yr radiol. scores were IgM rheumatoid factor positivity, DRB1*04 genes, ***pain*** score, and total radiol. score. Progression of joint damage was predicted by the ESR, IgM rheumatoid factor positivity, DRB1*04 genes,. . .

L6 ANSWER 30 OF 41 CAPLUS COPYRIGHT 2008 ACS on STN
AN 2000:128815 CAPLUS <<LOGINID::20080330>>
DN 132:164242
TI Molecular effect of physical exercise and stress on the skeletal muscle - the example of peripheral arterial occlusive disease
AU Steinacker, Jurgen M.; Lormes, Werner; Lehmann, Manfred; Liu, Yuefei
CS Abt. Sport- und Rehabilitationsmedizin, Medizinische Klinik und Poliklinik, Universitatsklinikum, Ulm, Germany
SO Deutsche Zeitschrift fuer Sportmedizin (2000), 51(1), 11-20
CODEN: DZSPD8; ISSN: 0344-5925
PB WWF Verlagsgesellschaft mbH
DT Journal; General Review
LA German
AB A review is given with 84 refs. The plasticity of skeletal muscle is high depending on stress and metab. Peripheral arterial occlusive disease (PAOD) leads to deficiency in perfusion, ***pain*** on exercise, and loss of phys. performance, and thus to important chains in peripheral perfusion and in the cellular reactions on the musculature. Chronic perfusion deficiency activates vasoconstrictive mechanisms, esp. the renin-angiotensin-system, with neg. effects on metab. and regulation of blood pressure. Phys. exercise leads to marked endogenous prodn. of vasodilating substances like NO, prostacycline, and bradykinin, and thus to improvement in peripheral perfusion, it induces local vascular growth factors (HIF, VEGF). Ischemia implies a high cellular stress with lack of energy, acidosis, free radicals. The prodn. of stress proteins (***heat*** ***shock*** ***protein*** ; HSP) is an important protective mechanism in the skeletal muscle in case of PAOD. HSP stabilizes the protein and gene structure of the cell, supporting by protein denaturation and protein synthesis as a "mol. chaperone". The myofibrillar proteins show a loss of rapid myosin heavy chains in PAOD and an expression of a slow, more energy-efficient muscle fiber type. Training and stress alter the gene expression via various pathways of cellular signal transduction. Walking training plays an important role in

the therapy of PAOD, whereby perfusion should be concurrently treated with medication or interventional means, and it improves the subjective well-being and exercise capacity of the patient.

RE.CNT 83 THERE ARE 83 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB . . . of skeletal muscle is high depending on stress and metab. Peripheral arterial occlusive disease (PAOD) leads to deficiency in perfusion, ***pain*** on exercise, and loss of phys. performance, and thus to important chains in peripheral perfusion and in the cellular reactions. . . (HIF, VEGF). Ischemia implies a high cellular stress with lack of energy, acidosis, free radicals. The prodn. of stress proteins (***heat*** ***shock*** ***protein*** ; HSP) is an important protective mechanism in the skeletal muscle in case of PAOD. HSP stabilizes the protein and gene. . .

IT ***Heat*** - ***shock*** ***proteins***
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(HSP in phys. exercise and stress effect on the skeletal muscle in peripheral arterial occlusive disease)

L6 ANSWER 31 OF 41 CAPLUS COPYRIGHT 2008 ACS on STN
AN 1999:600693 CAPLUS <<LOGINID::20080330>>
DN 132:116923
TI Effects of hyaluronic acid on cartilage degradation
AU Uebelhart, Daniel; Williams, James M.
CS Department of Clinical Neuroscience & Dermatology, Clinic of Rehabilitation, University Hospital-Beau-Sejour, Geneva, 1211/14, Switz.
SO Current Opinion in Rheumatology (1999), 11(5), 427-435
CODEN: CORHES; ISSN: 1040-8711
PB Lippincott Williams & Wilkins
DT Journal; General Review
LA English
AB A review, with 52 refs. Based on the published literature available so far, it appears that naturally derived hyaluronic acid (HA) and newer formulations available on the market belong to the pharmacol. class of slow-acting drugs for the treatment of osteoarthritis. These compds. seem to have the potential to modulate the painful symptoms of osteoarthritis as well as to improve the function of the osteoarthritis joint. Pos. clin. consequences are based on direct and indirect effects of viscosupplementation assocd. with a normalization of the rheol. properties of the osteoarthritic synovial fluid, decreased inflammation, and end-coating of the ***pain*** receptors in the osteoarthritis joint. Few in vivo data exist in humans to support the concept that HA formulations could have a structure-modifying effect on human osteoarthritis cartilage. Animal-based studies have demonstrated pos. effects of exogenous HA on ***pain*** in the joint, ***heat*** ***shock*** ***proteins*** , and in models of osteoarthritis. Although many promising effects of exogenous HA have been reported, there remains uncertainty as to the effectiveness of reversing cartilage injury and other manifestations of joint diseases with exogenous HA because of difficulties in interpreting and unifying results of these studies. This is due largely to differences of cartilage source in models of joint/cartilage injury, multiple end points, the controls employed, anal. techniques, and the mol. wt. of exogenous HA used. There exists a need for uniform agreement as to the choice of injury model, time points of the study, evaluation tools, and source and mol. wt. of the HA used if we are

to det. whether exogenous application of HA has a truly beneficial role in the reversal of cartilage injury.

RE.CNT 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

AB . . . viscosupplementation assocd. with a normalization of the rheol. properties of the osteoarthritic synovial fluid, decreased inflammation, and end-coating of the ***pain*** receptors in the osteoarthritis joint. Few in vivo data exist in humans to support the concept that HA formulations could have a structure-modifying effect on human osteoarthritis cartilage. Animal-based studies have demonstrated pos. effects of exogenous HA on ***pain*** in the joint, ***heat*** ***shock*** ***proteins*** , and in models of osteoarthritis. Although many promising effects of exogenous HA have been reported, there remains uncertainty as to. . .

L6 ANSWER 32 OF 41 MEDLINE on STN
AN 2007412554 MEDLINE <<LOGINID::20080330>>
DN PubMed ID: 17630143
TI The short-term effects of electrosurgical ablation on proinflammatory mediator production by intervertebral disc cells in tissue culture.
AU Rhyu Kee-Won; Walsh Andrew J L; O'Neill Conor W; Bradford David S; Lotz Jeffrey C
CS Department of Orthopaedic Surgery, University of California at San Francisco, 2100 Webster Street, Suite 110, San Francisco, CA 94115, USA.
SO The spine journal : official journal of the North American Spine Society, (2007 Jul-Aug) Vol. 7, No. 4, pp. 451-8. Electronic Publication: 2007-02-12.
Journal code: 101130732. ISSN: 1529-9430.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200709
ED Entered STN: 17 Jul 2007
Last Updated on STN: 19 Sep 2007
Entered Medline: 18 Sep 2007
AB BACKGROUND CONTEXT: Percutaneous discectomy can be performed by a variety of methods. One method, electrosurgical ablation, has been shown in a chronic animal model to alter the expression of inflammatory cytokines in degenerated discs. PURPOSE: To determine whether electrosurgical ablation has an acute direct effect on proinflammatory mediator production by disc cells. STUDY DESIGN: A short-term in vitro study using normal and interleukin (IL)-1alpha stimulated porcine disc cells cultured in alginate gel to evaluate the biochemical effects of electrosurgical ablation. METHODS: Porcine annulus and nucleus cells were embedded into alginate gels and cultured using control culture media or IL-1alpha-treated media for 6 days before ablation treatment. Treated gels were ablated by using a radiofrequency-based electrosurgical device for 5 seconds and cultured an additional 3 or 6 days. IL-1beta, IL-6, IL-8, tumor necrosis factor alpha (TNF-alpha), prostaglandin E2 (PGE2), nitric oxide (NO), and ***heat*** ***shock*** ***protein*** -70 (Hsp70) levels in culture medium were measured. Levels were normalized to DNA and compared between ablated and shams. RESULTS: For normal annulus cells, there were no significant changes in cytokine levels between ablation and sham groups. For normal nucleus cells, ablation produced significantly greater levels of IL-8 at 3 days and 6 days, Hsp70 at 3 days but not 6 days, and NO at 6 days. PGE2 was also increased at 3 days and 6 days but not

significantly. For IL-1-stimulated annulus cells, IL-6 and NO in the ablation group were decreased at 3 days relative to the control group. However, IL-6, IL-8, PGE2, and Hsp70 were significantly increased in the 6-day ablation group. For degenerated nucleus cells, IL-6, IL-8, and TNF-alpha were significantly decreased in the ablation group at both 3 days and 6 days. Ablation resulted in reduced PGE2 at 3 days but not 6 and reduced Hsp70 and NO at 6 days. CONCLUSIONS: The results show that electrosurgical ablation has an acute direct effect on proinflammatory mediator production by disc cells. The effect produced depends on disc cell phenotype, the mediator, and time. These direct biologic effects may be a mechanism of ***pain*** relief after percutaneous discectomy using electrosurgical ablation. However, the measured responses are limited to the short-term (1 week), and the existence of a prolonged effect remains to be determined.

AB . . . additional 3 or 6 days. IL-1beta, IL-6, IL-8, tumor necrosis factor alpha (TNF-alpha), prostaglandin E2 (PGE2), nitric oxide (NO), and ***heat*** ***shock*** ***protein*** -70 (Hsp70) levels in culture medium were measured. Levels were normalized to DNA and compared between ablated and shams. RESULTS: For. . . effect produced depends on disc cell phenotype, the mediator, and time. These direct biologic effects may be a mechanism of ***pain*** relief after percutaneous discectomy using electrosurgical ablation. However, the measured responses are limited to the short-term (1 week), and the. . .

CT Animals
Catheter Ablation
Cells, Cultured
Cytokines: AI, antagonists & inhibitors
*Cytokines: BI, biosynthesis
Dinoprostone: BI, biosynthesis
*Electrosurgery
Electrosurgery: MT, methods
*** HSP72 Heat-Shock Proteins: BI, biosynthesis***
Inflammation Mediators: AI, antagonists & inhibitors
*Inflammation Mediators: ME, metabolism
Interleukin-1alpha: PD, pharmacology
Intervertebral Disk:. . .

CN 0 (Cytokines); 0 (HSP72 ***Heat*** - ***Shock*** ***Proteins***); 0 (Inflammation Mediators); 0 (Interleukin-1alpha)

L6 ANSWER 33 OF 41 MEDLINE on STN
AN 2004528679 MEDLINE <<LOGINID::20080330>>
DN PubMed ID: 15349045
TI Ocular cicatricial pemphigoid review.
AU Foster C Stephen; Sainz De La Maza Maite
CS Department of Ophthalmology, Harvard Medical School, Immunology and Uveitis Service, Massachusetts Eye and Ear Infirmary, Boston, Massachusetts 02114, USA.. fosters@uveitis.org
SO Current opinion in allergy and clinical immunology, (2004 Oct) Vol. 4, No. 5, pp. 435-9. Ref: 27
Journal code: 100936359. ISSN: 1528-4050.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA English
FS Priority Journals
EM 200503
ED Entered STN: 26 Oct 2004

Last Updated on STN: 11 Mar 2005
Entered Medline: 10 Mar 2005

AB PURPOSE OF REVIEW: Ocular mucous membrane pemphigoid produces progressive cicatrizing conjunctivitis; this can result in scarring of the conjunctiva and cornea. RECENT FINDINGS: Increased expression of macrophage-colony-stimulating factor, collagen-binding ***heat*** ***shock*** ***protein*** 47, transforming growth factor-beta1, IL-4, IL-5, and macrophage migration inhibitory factor may enhance both conjunctival inflammation and conjunctival scarring. Recent developments on mucous membrane pemphigoid medical therapy include the efficacious effect of daclizumab, intravenous immunoglobulin therapy, and methotrexate. Subconjunctival mitomycin has proved not to be efficacious in controlling long-term, conjunctival inflammation and scarring. The Boston scleral lens enhances vision, reduces the disabling ocular ***pain*** and photophobia, and helps to heal persistent epithelial defects, reducing recurrence of defects. Recent developments on mucous membrane pemphigoid surgical therapy include keratolimbal allografts and amniotic membrane transplantation, with or without penetrating keratoplasty for ocular surface reconstruction in total stem cell deficiency. The prognosis is strongly influenced by preoperative conditions such as tear function and functional external ocular adnexae, and by postoperative conditions such as persistent inflammation, severe dry eye, or rejection of the keratolimbal allograft. Some authors find efficacious the use of amniotic membrane transplantation for reconstruction of the conjunctival fornices provided systemic immunosuppression is pre- and post-operatively used. SUMMARY: The use of daclizumab or intravenous immunoglobulin therapy, the better selection of candidates for surgical interventions, and the better pre- and post-operative management of keratolimbal allograft and amniotic membrane transplantation may improve visual rehabilitation in the patients with ocular mucous membrane pemphigoid.

AB . . . cicatrizing conjunctivitis; this can result in scarring of the conjunctiva and cornea. RECENT FINDINGS: Increased expression of macrophage-colony-stimulating factor, collagen-binding ***heat*** ***shock*** ***protein*** 47, transforming growth factor-beta1, IL-4, IL-5, and macrophage migration inhibitory factor may enhance both conjunctival inflammation and conjunctival scarring. Recent. . . to be efficacious in controlling long-term, conjunctival inflammation and scarring. The Boston scleral lens enhances vision, reduces the disabling ocular ***pain*** and photophobia, and helps to heal persistent epithelial defects, reducing recurrence of defects. Recent developments on mucous membrane pemphigoid surgical. . .

L6 ANSWER 34 OF 41 MEDLINE on STN
AN 2003485960 MEDLINE <<LOGINID::20080330>>
DN PubMed ID: 14563459
TI Musculoskeletal manifestations in cystic fibrosis.
AU Botton Estelle; Saraux Alain; Laselve Hermine; Jousse Sandrine; Le Goff Paul
CS Service de rhumatologie, Hopital de la cavale blanche, CHU Brest, 29609 Brest cedex, France.
SO Joint, bone, spine : revue du rhumatisme, (2003 Sep) Vol. 70, No. 5, pp. 327-35. Ref: 131
Journal code: 100938016. ISSN: 1297-319X.
CY France
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA English

FS Priority Journals
EM 200403
ED Entered STN: 18 Oct 2003
Last Updated on STN: 17 Mar 2004
Entered Medline: 16 Mar 2004
AB Although bone and joint manifestations are common in children with cystic fibrosis (CF), they have received little attention in adults. As compared to healthy individuals, bone mineral density is low, even with calcium intakes greater than 1500 mg/d. Nevertheless, calcium and phosphate levels in blood and urine are often normal, and vitamin D levels vary. Short stature with a low body mass index and central hypogonadism are the rule in these patients. Fractures and kyphosis are often reported. CF arthropathy occurs in 2-8.5% of patients. Arthritis develops, and there may be skin eruptions. Non-steroidal antiinflammatory drug therapy is effective. Hypertrophic osteoarthropathy associated with respiratory failure is present in 2-7% of patients. Rheumatoid arthritis, spondyloarthropathies, sarcoidosis, and amyloidosis have been reported in association with CF. Knee ***pain*** due to patellofemoral syndrome, quinolone-induced arthropathy, and mechanical back ***pain*** have been described. Rheumatoid factor titers are higher than in healthy controls, particularly in patients with episodic arthritis. No data are available on antiperinuclear factor or antikeratin antibody titers. Tests for antinuclear antibody are usually negative. Circulating immune complex levels and antibodies to ***heat*** ***shock*** ***proteins*** may be elevated. Antineutrophil cytoplasmic antibody of the bactericidal/permeability-increasing protein (BPI) or azurocidin (AZ) type has been reported, often in high titers (up to 40%).
AB . . . is present in 2-7% of patients. Rheumatoid arthritis, spondyloarthropathies, sarcoidosis, and amyloidosis have been reported in association with CF. Knee ***pain*** due to patellofemoral syndrome, quinolone-induced arthropathy, and mechanical back ***pain*** have been described. Rheumatoid factor titers are higher than in healthy controls, particularly in patients with episodic arthritis. No data. . . antiperinuclear factor or antikeratin antibody titers. Tests for antinuclear antibody are usually negative. Circulating immune complex levels and antibodies to ***heat*** ***shock*** ***proteins*** may be elevated. Antineutrophil cytoplasmic antibody of the bactericidal/permeability-increasing protein (BPI) or azurocidin (AZ) type has been reported, often in. . .

L6 ANSWER 35 OF 41 MEDLINE on STN
AN 2002711779 MEDLINE <<LOGINID::20080330>>
DN PubMed ID: 12473980
TI Ischemic preconditioning. Experimental facts and clinical perspective.
AU Post H; Heusch G
CS Institute of Pathophysiology, University of Essen, Essen, Germany.
SO Minerva cardioangiologica, (2002 Dec) Vol. 50, No. 6, pp. 569-605. Ref: 409
Journal code: 0400725. ISSN: 0026-4725.
CY Italy
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA English
FS Priority Journals
EM 200309
ED Entered STN: 17 Dec 2002
Last Updated on STN: 30 Sep 2003

Entered Medline: 29 Sep 2003

AB Brief periods of non-lethal ischemia and reperfusion render the myocardium more resistant to subsequent ischemia. This adaption occurs in a biphasic pattern: the first being active immediately and lasting for 2-3 hrs (early preconditioning), the second starting at 24 hrs until 72 hrs after the initial ischemia (delayed preconditioning) and requiring genomic activation with de novo protein synthesis. Early preconditioning is more potent than delayed preconditioning in reducing infarct size; delayed preconditioning also attenuates myocardial stunning. Early preconditioning depends on the ischemia-induced release of adenosine and opioids and, to a lesser degree, also bradykinin and prostaglandins. These molecules activate G-protein coupled receptors, initiate the activation of KATP channels and generation of oxygen radicals, and stimulate a series of protein kinases with essential roles for protein kinase C, tyrosine kinases and members of the MAP kinase family. Delayed preconditioning is triggered by a similar sequence of events, but in addition essentially depends on eNOS-derived NO. Both early and pharmacological preconditioning can be pharmacologically mimicked by exogenous adenosine, opioids, NO and activators of protein kinase C. Newly synthetized proteins associated with delayed preconditioning comprise iNOS, COX-2, manganese superoxide dismutase and possibly ***heat*** ***shock*** ***proteins*** . The final mechanism of protection by preconditioning is yet unknown; energy metabolism, KATP channels, the sodium-proton exchanger, stabilisation of the cytoskeleton and volume regulation will be discussed. For ethical reasons, evidence for ischemic preconditioning in humans is hard to provide. Clinical findings that parallel experimental ischemic preconditioning are reduced ST-segment elevation and ***pain*** during repetitive PTCA or exercise tests, a better prognosis of patients in whom myocardial infarction was preceded by angina, and reduced serum markers of myocardial necrosis after preconditioning protocols during cardiac surgery with cardiac arrest. The most promising approach to apply principles of ischemic preconditioning therapeutically appears to be the pharmacological recruitment of delayed protection, as recently demonstrated with intravenous nitroglycerine in patients undergoing PTCA 24 hrs later.

AB . . . activators of protein kinase C. Newly synthetized proteins associated with delayed preconditioning comprise iNOS, COX-2, manganese superoxide dismutase and possibly ***heat*** ***shock*** ***proteins*** . The final mechanism of protection by preconditioning is

yet unknown; energy metabolism, KATP channels, the sodium-proton exchanger, stabilisation of the. . . ischemic preconditioning in humans is hard to provide. Clinical findings that parallel experimental ischemic preconditioning are reduced ST-segment elevation and ***pain*** during repetitive PTCA or exercise tests, a better prognosis of patients in whom myocardial infarction was preceded by angina, and. . .

L6 ANSWER 36 OF 41 MEDLINE on STN

AN 2002404306 MEDLINE <<LOGINID::20080330>>

DN PubMed ID: 12153633

TI Influence of implantation interval on the long-term biocompatibility of surgical mesh.

AU Klosterhalfen B; Junge K; Hermanns B; Klinge U

CS Institute of Pathology, German Centre of Excellence for Biomaterial and Implant Pathology, Interdisciplinary Centre of Clinical Science BIOMAT, Rhenish Westfalian Technical High School-Aachen, Aachen, Germany.. klosterhalfen@pat.rwth-aachen.de

SO The British journal of surgery, (2002 Aug) Vol. 89, No. 8, pp. 1043-8.
Journal code: 0372553. ISSN: 0007-1323.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 200209
ED Entered STN: 3 Aug 2002
Last Updated on STN: 11 Sep 2002
Entered Medline: 10 Sep 2002
AB BACKGROUND: The aim was to study the long-term tissue response to polypropylene mesh. METHODS: This was a retrieval study that investigated 76 polypropylene meshes with a median implantation interval of 18 (range 2-180) months. Mesh was explanted following hernia recurrence, infection or ***pain*** . The median implantation interval was 20 (range 4-180) months in the recurrence group, 30 (range 5-48) months in the ***pain*** group and 10 (range 2-56) months in the infection group (P < 0.05, infection versus ***pain*** or recurrence). The inflammatory response was determined by immunohistochemistry of macrophages (CD68), polymorphonuclear granulocytes (CD15) and T and B lymphocytes (CD3 and CD20). The cell turnover within the interface mesh fibre-recipient tissue was measured by TUNEL for apoptosis or DNA strand breaks, Ki67 for cell proliferation and ***heat*** - ***shock*** ***protein*** (HSP) 70 for cell stress. RESULTS: With the exception of HSP-70, levels of all variables decreased over time. Sex, age, type of previous operation or location of the mesh did not have a significant influence. CONCLUSION: Long-term incorporated polypropylene mesh in humans has a more favourable tissue response with increasing implantation interval.
AB . . . polypropylene meshes with a median implantation interval of 18 (range 2-180) months. Mesh was explanted following hernia recurrence, infection or ***pain*** . The median implantation interval was 20 (range 4-180) months in the recurrence group, 30 (range 5-48) months in the ***pain*** group and 10 (range 2-56) months in the infection group (P < 0.05, infection versus ***pain*** or recurrence). The inflammatory response was determined by immunohistochemistry of macrophages (CD68), polymorphonuclear granulocytes (CD15) and T and B lymphocytes. . . the interface mesh fibre-recipient tissue was measured by TUNEL for apoptosis or DNA strand breaks, Ki67 for cell proliferation and ***heat*** - ***shock*** ***protein*** (HSP) 70 for cell stress. RESULTS: With the exception of HSP-70, levels of all variables decreased over time. Sex, age,. . .

L6 ANSWER 37 OF 41 MEDLINE on STN
AN 2001149154 MEDLINE <<LOGINID::20080330>>
DN PubMed ID: 11214479
TI [The modern approach to wound treatment].
Savremeni pristup tretmanu rana.
AU Komarcevic A
CS Institut za zdravstvenu zastitu dece i omladine Klinika za decju hirurgiju, Medicinski fakultet, Novi Sad.. komarac@Eunet.yu
SO Medicinski pregled, (2000 Jul-Aug) Vol. 53, No. 7-8, pp. 363-8. Ref: 52
Journal code: 2985249R. ISSN: 0025-8105.
CY Yugoslavia
DT (ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)

LA Croatian
FS Priority Journals
EM 200103
ED Entered STN: 4 Apr 2001
Last Updated on STN: 4 Apr 2001
Entered Medline: 15 Mar 2001
AB INTRODUCTION: Wound healing is a complex process involving interactions among a variety of different cell types. The normal wound repair process consists of three phases--inflammation, proliferation, and remodeling that occur in a predictable series of cellular and biochemical events. Wounds are classified according to various criteria: etiology, lasting, morphological characteristics, communications with solid or hollow organs, the degree of contamination. In the last few years many authors use the Color Code Concept, which classifies wounds as red, yellow and black wounds. This paper presents conventional methods of local wound treatment (mechanical cleansing, disinfection with antiseptic solutions, wound debridement--surgical, biological and autolytic; wound closure, topical antibiotic treatment, dressing), as well as general measures (sedation, antitetanous and antibiotic protection, preoperative evaluation and correction of malnutrition, vasoconstriction, hyperglycemia and steroid use, appropriate surgical technique, and postoperative prevention of vasoconstriction through ***pain*** relief, warming and adequate volume resuscitation). THE ROLE OF PHYSIOLOGICAL FACTORS AND ANTIMICROBIAL AGENTS IN WOUND HEALING: Growth factors play a role in cell division, migration, differentiation, protein expression, enzyme production and have a potential ability to heal wounds by stimulating angiogenesis and cellular proliferation, affecting the production and the degradation of the extracellular matrix, and by being chemotactic for inflammatory cells and fibroblasts. There are seven major families of growth factors: epidermal growth factor (EGF), transforming growth factor-beta (TGF-beta), insulin-like growth factor (IGF), platelet-derived growth factor (PDGF), fibroblast growth factor (FGF), interleukins (ILs), and colony-stimulating factor (CSF). Acute wounds contain many growth factors that play a crucial role in the initial phases of wound healing. The events of early wound healing reflect a finely balanced environment leading to uncomplicated and rapid wound healing. Chronic wounds, for many reasons, have lost this fine balance. Multiple studies have evaluated the effect that exogenously applied growth factors have on the healing of chronic wounds. In the study conducted by Knighton and colleagues, topical application of mixture of various growth factors (PDGF, TGF-beta, PDAF, PF4, PDEGF) demonstrated increased wound healing over controls. Brown and associates demonstrated a decrease in skin graft donor site healing time of 1 day using topically applied EGF. Herndon and ass. used systemic growth hormone in burned children and reduction in healing time made a significant clinical difference by allowing earlier wound coverage and decreasing the duration of hospitalization. The TGF family of growth factors is believed to be primarily responsible for excessive scar formation, especially the beta 1 and beta 2 isoforms. TGF-beta 3 isoform has recently been described and may have an inhibitory function on scar formation by being a natural antagonist to the TGF-beta 1 and TGF-beta 2 isoforms. Cytokines, especially interferon-alpha (INF-alpha), INF-alpha, and INF-alpha 2b, may also reduce scar formation. These cytokines decrease the proliferation rate of fibroblasts and reduce the rate of collagen and fibronectin synthesis by reducing the production of mRNA. Expression of nitric oxide synthase (NOS) and ***heat*** ***shock*** ***proteins*** (HSP) have an important role in wound healing, as well as trace elements (zinc, copper, manganese).

Applications of some drugs (antioxidants--asiaticoside, vitamin E and ascorbic acid; calcium D-pantothenate, exogenous fibronectin; antileprosy drugs--oil of hydnocarpus; alcoholic extract of yeast) accelerate wound healing. Thymic peptide thymosin beta 4 (T beta 4R) topically applicated, increases collagen deposition and angiogenesis and stimulates keratinocyte migration. Thymosin alpha 1 (T alpha 1R), peptide isolated from the thymus, is a potent chemoattractant which accelerates angiogenesis and wound healing. On the contrary, steroid drugs, hemorrhage and denervation of wounds have negative effect on the healing process.

AB . . . preoperative evaluation and correction of malnutrition, vasoconstriction, hyperglycemia and steroid use, appropriate surgical technique, and postoperative prevention of vasoconstriction through ***pain*** relief, warming and adequate volume resuscitation). THE ROLE OF PHYSIOLOGICAL FACTORS AND ANTIMICROBIAL AGENTS IN WOUND HEALING: Growth factors play. . . the rate of collagen and fibronectin synthesis by reducing the production of mRNA. Expression of nitric oxide synthase (NOS) and ***heat*** ***shock*** ***proteins*** (HSP) have an important role in wound healing, as well as trace elements (zinc, copper, manganese). Applications of some drugs. . .

L6 ANSWER 38 OF 41 MEDLINE on STN

AN 1999297883 MEDLINE <<LOGINID::20080330>>

DN PubMed ID: 10371473

TI Infertility following pelvic inflammatory disease.

AU Pavletic A J; Wolner-Hanssen P; Paavonen J; Hawes S E; Eschenbach D A

CS Department of Family Medicine, University of Nebraska Medical Center, Omaha 68198-3075, USA.

NC AI 24756 (United States NIAID)
AI 33118 (United States NIAID)

SO Infectious diseases in obstetrics and gynecology, (1999) Vol. 7, No. 3, pp. 145-52.
Journal code: 9318481. ISSN: 1064-7449.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)

LA English

FS Priority Journals

EM 199907

ED Entered STN: 6 Aug 1999
Last Updated on STN: 6 Aug 1999
Entered Medline: 29 Jul 1999

AB OBJECTIVE: To assess the frequency of infertility after pelvic inflammatory disease (PID) and factors important in postinfectious tubal damage in an urban population at high risk for sexually transmitted diseases. METHODS: From a cohort of 213 women with PID documented by laparoscopy and/or endometrial biopsy, 58 women (27% of the initial cohort) were interviewed by phone 2 to 9 years after an index episode of PID. Data regarding the initial history, physical examination, microbiology, laparoscopic, and serologic findings, and data concerning interval contraception, subsequent pregnancy, subsequent infection, and chronic pelvic ***pain*** were compared among those with and without infertility at follow up. RESULTS: Nineteen (40%) of the 48 women not using contraception were involuntarily infertile after the index episode of PID. Compared with those who had an interval pregnancy, infertile women were older (P = 0.02), more likely to have a history of infertility prior to the index episode of PID (P = 0.001), and were more likely to

have occluded or partially occluded fallopian tubes (P = 0.03), peritubal adhesions (P = 0.007), or perihepatic adhesions (P = 0.02) seen by laparoscopy performed during the index episode. Surprisingly, recovery of Chlamydia trachomatis was negatively related to infertility (P = 0.001), although a similar proportion of both groups had chlamydia immunoglobulin M antibody (40% vs. 31%). Chlamydia ***heat*** ***shock*** ***protein*** was weakly related to infertility (P = 0.08). The isolation of Neisseria gonorrhoeae was not significantly different between groups (53% vs. 57%). CONCLUSIONS: The high rate of postinfection infertility found was probably related to a combination of tubal damage before and during the index episode of PID. Prevention of recurrent PID and better understanding of the pathophysiology of postinfection tubal damage (which may differ between chlamydia and gonorrhea) is needed to develop more effective strategies to reduce permanent tubal damage.

AB . . . history, physical examination, microbiology, laparoscopic, and serologic findings, and data concerning interval contraception, subsequent pregnancy, subsequent infection, and chronic pelvic ***pain*** were compared among those with and without infertility at follow up. RESULTS: Nineteen (40%) of the 48 women not using. . . infertility (P = 0.001), although a similar proportion of both groups had chlamydia immunoglobulin M antibody (40% vs. 31%). Chlamydia ***heat*** ***shock*** ***protein*** was weakly related to infertility (P = 0.08). The isolation of Neisseria gonorrhoeae was not significantly different between groups (53%. . .

L6 ANSWER 39 OF 41 MEDLINE on STN
AN 1998159374 MEDLINE <<LOGINID::20080330>>
DN PubMed ID: 9497939
TI Analysis of ***heat*** ***shock*** ***proteins*** and cytokines expressed during early stages of osteoarthritis in a mouse model.
AU Takahashi K; Kubo T; Goomer R S; Amiel D; Kobayashi K; Imanishi J; Teshima R; Hirasawa Y
CS Department of Orthopaedic Surgery, Kyoto Prefectural University of Medicine, Japan.
NC AR 07484 (United States NIAMS)
SO Osteoarthritis and cartilage / OARS, Osteoarthritis Research Society, (1997 Sep) Vol. 5, No. 5, pp. 321-9.
Journal code: 9305697. ISSN: 1063-4584.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LA English
FS Priority Journals
EM 199803
ED Entered STN: 26 Mar 1998
Last Updated on STN: 26 Mar 1998
Entered Medline: 19 Mar 1998

AB OBJECTIVE: Osteoarthritis (OA) is a debilitating disease of the joints. The joints of affected individuals are characterized by a progressive degeneration of articular cartilage leading to inflammation and ***pain*** . The expression of ***heat*** ***shock*** ***proteins*** (HSPs) is a ubiquitous self-protective mechanism of all cells under stress, furthermore, the synovium of osteoarthritic individuals contains high levels of cytokines. This study seeks to establish the role of HSPs and cytokines in OA. METHODS: We have

investigated the presence of HSPs and cytokines in articular cartilage during early stages of OA in a mouse that is known to develop spontaneous OA lesions (C57 black mouse). The articular cartilage from closely related mice (C57BL/6) was used as control. Messenger RNAs (mRNAs) for HSPs (HSP32, HSP47, HSP60, HSP70, HSP84 and HSP86) and cytokines [interleukin-1 beta (IL-1 beta), interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-alpha) and interferon-gamma (IFN-gamma)] were detected by reverse transcription-polymerase chain reaction (RT-PCR). RESULTS: The mRNA levels of HSP47, HSP70, HSP86, IL-6, and IFN-gamma were up-regulated in the cartilage of C57 black mice, whereas, the level of expression of HSP32, HSP60, HSP84 and IL-1 beta remained unchanged. Furthermore, the expression of IL-1 beta, IL-6, TNF-alpha and IFN-gamma mRNA was associated with expression of HSP60, HSP47, HSP70 and HSP70/HSP86 mRNA, respectively. CONCLUSIONS: The findings in this study suggest that chondrocytes are conditioned under non-physiological stress during early stages of OA, In addition, among HSPs, HSP70 was associated with two different highly expressed cytokines in C57 black mice, indicating the possible role of HSP70 as a characteristic indicator of early stage of OA.

TI Analysis of ***heat*** ***shock*** ***proteins*** and cytokines expressed during early stages of osteoarthritis in a mouse model.

AB . . . the joints. The joints of affected individuals are characterized by a progressive degeneration of articular cartilage leading to inflammation and ***pain*** . The expression of ***heat*** ***shock*** ***proteins*** (HSPs) is a ubiquitous self-protective mechanism of all cells under stress, furthermore, the synovium of osteoarthritic individuals contains high levels. . .

CT Check Tags: Male
Animals
Cartilage, Articular: ME, metabolism
Cytokines: GE, genetics
*Cytokines: ME, metabolism
Disease Models, Animal
Gene Expression
*** Heat-Shock Proteins: GE, genetics***
****Heat-Shock Proteins: ME, metabolism***
Knee Joint: ME, metabolism
Knee Joint: PA, pathology
Mice
Mice, Inbred C57BL
*Osteoarthritis: ME, metabolism
Osteoarthritis: PA,. . .

CN 0 (Cytokines); 0 (***Heat*** - ***Shock*** ***Proteins***); 0 (RNA, Messenger)

L6 ANSWER 40 OF 41 MEDLINE on STN

AN 94162593 MEDLINE <<LOGINID::20080330>>

DN PubMed ID: 8118005

TI [The antimutagenic effect of adaptation to stress]. Antimutagennyi effekt adaptatsii k stressu.

AU Meerson F Z; Kulakova A V; Saltykova V A

SO Biulleten' eksperimental'noi biologii i meditsiny, (1993 Sep) Vol. 116, No. 9, pp. 292-5.
Journal code: 0370627. ISSN: 0365-9615.

CY RUSSIA: Russian Federation

DT (COMPARATIVE STUDY)
(ENGLISH ABSTRACT)

Journal; Article; (JOURNAL ARTICLE)
LA Russian
FS Priority Journals
EM 199404
ED Entered STN: 12 Apr 1994
Last Updated on STN: 12 Apr 1994
Entered Medline: 6 Apr 1994
AB C57BL mice were adapted to moderate periodic hypoxia and repeated electric ***pain*** stresses of limited intensity. These animals adapted to each of the factors and control animals were given the potent mutagen, free radical oxidation activator dioxidine in a single dose of 300 mg/kg. Dioxidine administered to unadapted animals resulted in chromosomal aberrations in 11% of stem bone marrow cells mainly due to the appearance of single and multiple chromosomes. Preadaptation to stress decreased the number of these dioxidine-induced chromosomal aberrations nearly twice. Adaptation to periodic hypoxia had no defensive action. As previously shown, adaptation to repeated stresses leads to the accumulation of ***heat*** - ***shock*** ***proteins*** (HSP) in the cellular nuclei of animals and prevents the degradation of isolated nuclei when single-chain DNA is added. Adaptation to hypoxia does not cause nuclear accumulation of HSP or prevents their degradation when unicellular DNA is supplemented. This suggests that the antimutagenic effect of stress adaptation is likely to be accounted for by the stabilizing action of HSP.
AB C57BL mice were adapted to moderate periodic hypoxia and repeated electric ***pain*** stresses of limited intensity. These animals adapted to each of the factors and control animals were given the potent mutagen,. . . Adaptation to periodic hypoxia had no defensive action. As previously shown, adaptation to repeated stresses leads to the accumulation of ***heat*** - ***shock*** ***proteins*** (HSP) in the cellular nuclei of animals and prevents the degradation of isolated nuclei when single-chain DNA is added. Adaptation. . .
CT . . .
Hematopoietic Stem Cells: DE, drug effects
Mice
Mice, Inbred C57BL
Mutagenesis: DE, drug effects
*Mutagenesis: PH, physiology
Mutagens: PD, pharmacology
*** Pain: GE, genetics***
*** Pain: PP, physiopathology***
Periodicity
Quinoxalines: PD, pharmacology
Stress: GE, genetics
*Stress: PP, physiopathology

L6 ANSWER 41 OF 41 MEDLINE on STN
AN 94001368 MEDLINE <<LOGINID::20080330>>
DN PubMed ID: 7691139
TI Juvenile rheumatoid arthritis.
AU Tucker L B
CS Division of Pediatric Rheumatology, Floating Hospital for Children, New England Medical Center, Boston MA 02111.
SO Current opinion in rheumatology, (1993 Sep) Vol. 5, No. 5, pp. 619-28.
Ref: 46
Journal code: 9000851. ISSN: 1040-8711.

CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LA English
FS Priority Journals
EM 199311
ED Entered STN: 17 Jan 1994
Last Updated on STN: 3 Feb 1997
Entered Medline: 24 Nov 1993
AB The etiology and pathogenesis of juvenile rheumatoid arthritis remains unknown; however, research using new techniques is revealing information on the roles of immunogenetics, cellular immunity, and humoral immunity in these disorders. Interest continues in infection as a potential trigger of juvenile rheumatoid arthritis, as reactivity to infectious agents in synovial lymphocytes is sought. Reactivity to ***heat*** - ***shock*** ***proteins*** suggests a pathogenetic role for this class of proteins as well. Careful analysis of outcome in children with systemic-onset juvenile rheumatoid arthritis has identified clinical features that may predict later clinical course; in related work, investigators have not been able to identify such features to predict the outcome of children with pauciarticular juvenile rheumatoid arthritis. Juvenile rheumatoid arthritis has a broad impact on the lives of patients and their families, and appropriate assessment and management of such problems as chronic ***pain*** , vocational readiness, and family financing have been addressed over the past year.
AB . . . as a potential trigger of juvenile rheumatoid arthritis, as reactivity to infectious agents in synovial lymphocytes is sought. Reactivity to ***heat*** - ***shock*** ***proteins*** suggests a pathogenetic role for this class of proteins as well. Careful analysis of outcome in children with systemic-onset juvenile. . . broad impact on the lives of patients and their families, and appropriate assessment and management of such problems as chronic ***pain*** , vocational readiness, and family financing have been addressed over the past year.
CT . . . Rheumatoid: IM, immunology
Arthritis, Reactive
Autoantibodies: IM, immunology
B-Lymphocytes: IM, immunology
Bacterial Infections
Bacterial Proteins: IM, immunology
Child
Chronic Disease
*** Heat-Shock Proteins: IM, immunology***
Humans
Iridocyclitis
Major Histocompatibility Complex
Mycobacterium Infections
*** Pain Measurement***
Parvoviridae Infections
Prognosis
CN 0 (Antigens, CD); 0 (Antigens, CD5); 0 (Autoantibodies); 0 (Bacterial Proteins); 0 (***Heat*** - ***Shock*** ***Proteins***)